THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—45TH YEAR SYDNEY, SATURDAY, APRIL 5, 1958 No. 14

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EARLY SETTLEMENT IN NORTHERN AUSTRALIA.

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PART II: PORT ESSINGTON.

IN spite of the unfortunate results of the two earlier attempts to found a settlement on the north coast of Australia, there was a strong body of opinion, particularly in official circles in London, that the rapid development of Sydney, and its progressively increasing trade with the East (the tonnage of vessels passing through Torres Straits increased from 4700 in 1832 to 14,950 in 1838), made such an establishment more and more essential. Its main objects would be: (*a*) to test adequately the possibilities of trade with the Malay Archipelago; (*b*) to develop tropical agriculture on a commercial scale; (*c*) by a show of naval power to prevent a recurrence of disasters such as those of the *Lady Nelson* and the *Stedcombe*; (*b*) to establish a port of refuge for shipwrecked personnel, since increased shipping through Torres Straits had brought more wrecks with such occasional disastrous results as that of the *Charles Eaton*,[1] and some haven for their relief was an urgent necessity. The real deciding factor, however, was a rumour that the French were preparing a force at Toulon to make a landing in the area. As to the actual site of the settlement, the enthusiasm of King and Bremer for the beautiful harbour of Port Essington made its selection inevitable, although to them its main advantage was the excellent anchorage it afforded.

Once again Captain (now Sir Gordon) Bremer was appointed to organize the settlement. He left Sydney on September 18, 1838, in H.M.S. *Alligator*, accompanied by Lieutenant Owen Stanley (a name destined to become very familiar to members of the Australian Imperial Force in

[1] The *Charles Eaton* left Sydney for Singapore on July 29, 1834, and was wrecked on Booby Island in Torres Straits in mid-August. Five members of the crew contrived to escape to an island near Timor, whence after a year they were allowed to proceed to Amboyna (it is suggested that only two of them lived to reach England). Of the six passengers and 23 crew, all the others were killed (including the surgeon, F. Grant), with the exception of one of the ship's apprentices and the small son of one of the passengers, Captain D'Oyley. They were kept captive for some months, when they were bought for a bunch of bananas each by natives from Murray Island, who treated them quite kindly until they were rescued on June 19, 1836, by the *Isabella* (Captain Lewis).

New Guinea) in H.M.S. *Britomart* and the storeship *Orontes*. They arrived in Port Essington on October 27. Prefabricated huts, a hospital and a church with capacity for 300, provided by the Society for the Propagation of the Gospel (there was no padre to function in it), were included in the equipment. The Commandant was Captain John Macarthur; the medical officer was Assistant Surgeon Frederick Whipple: the other personnel were marines, some 50 in all. The settlement, 16 miles from the entrance, was named Victoria in honour of the newly crowned Queen, but few, other than Macarthur, ever used the name. The erection of the buildings went on apace, and rapid progress was made with the garden. On December 16, the *Orontes*, sailing out of the port, ran aground on a shoal, which thereafter bore her name, and became a total wreck. Bremer at least had no misgivings as to the success of the undertaking. In 1839 he wrote:

> Nor can I entertain a doubt but that with the due encouragement it will receive from Home, its admirable geographical position will excite attention, its capabilities for mercantile purposes be appreciated, and its soil, which evidently will produce the most valuable articles, be speedily and successfully cultivated.

Unfortunately "the due encouragement" was not forthcoming, and his optimism proved to be as much at fault in this instance as it had been earlier in regard to Fort Dundas. In April, 1839, the natives told Bremer that there were two large ships at Raffles Bay. Investigation proved these to be the French vessels *L'Astrolabe* and *La Zélée*, commanded by Capitaine Dumont D'Urville. Their presence was at first regarded with some misgiving, but amicable relations were established, and they were invited to visit the settlement, where they remained for a short period.

In July, 1839, the *Alligator* returned to Port Jackson; when she arrived there it was necessary to admit to the General Hospital 25 of her crew, who were suffering from scurvy. This caused grave anxiety about the health of the garrison and the crew of the *Britomart*; the *Pelorus* was dispatched forthwith with the necessary supplies for their relief. However, the garrison were not in need of any assistance, for they had a fair supply of vegetables and had managed to maintain excellent health.

This standard of health continued, in general, through the early years of the settlement, although suitable diet was not always available, and some of the marines showed slight manifestations of scurvy. Most of the community suffered from some degree of ophthalmia, and flies, sandflies and mosquitoes were very troublesome. A hurricane in November, 1839, did much damage—it destroyed many of the fruit trees and a quantity of stores, it unroofed huts and seriously damaged the hospital, the jetty and the church. The *Pelorus* was driven ashore and 12 of the crew were drowned. The vessel itself was badly strained and, although it was repaired, its efficiency was greatly reduced, and it was eventually "sold out of the Service" and replaced by H.M.S. *Royalist*. Throughout the life of the settlement, hurricanes occurred at irregular intervals, and invariably did great damage to buildings, trees and crops. There were also occasional earthquakes, which caused only minor damage.

The marines were encouraged to develop gardens round their own huts, and there was, in addition, a communal garden, which in early days promised well, with vigorous growth of fruit trees and vegetables. However, in the long run, because of too much rain, or too little, excessive heat, hurricanes, destruction by rats or thieving by natives, production fell far below requirements.

The garrison had to provide its own entertainment, which included on August 24, 1839, the performance of a play "Cheap Living", in which all parts, male and female, were taken by marines. Owen Stanley was stage manager, and

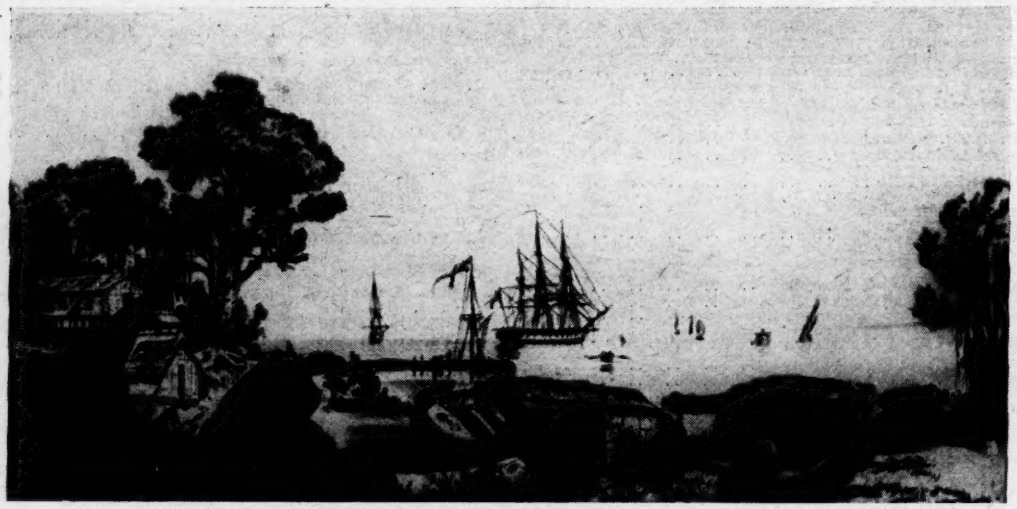

FIGURE I.
The harbour, Port Essington.

Even as early as 1840, there was evidence of the activity of termites in the various buildings, which now consisted of a hospital, a church and 24 cottages. The original hospital damaged by hurricane was remodelled as a storeroom, and a new hospital was built on a stone foundation; it contained four good-sized wards, a dispensary and quarters for the medical officer.

Development.

The main requirement for the new settlement was increased population; whites with money to invest, and Malays or Chinese to do the laborious work, for which it was soon realized Europeans were unfitted. Gipps and Bremer proposed to arrange for the sale of land, but the Home Government would not agree. Bremer then drew up a scheme for the lease of land for seven years: half-acre blocks in the settlement, and five-acre blocks in rural areas. But it was soon evident that people who could readily buy land in Sydney would not pay money for a permissive occupancy in Port Essington. The Colonial Land and Emigration Commissioners, who in fact controlled the settlement, endeavoured to interest merchants in London in the scheme, but they were not to be tempted: they still remembered only too well the fate of *Lady Nelson* and

Stedcombe. In Sydney there was a serious commercial depression, and traders there were far too busy conserving their existing assets to engage in a new venture which was largely experimental. Reports from Bremer and Macarthur were always encouraging, but those from other sources were much less optimistic. In 1840, the commissioners were asked by Lord John Russell to advise about the possibility of maintaining the settlement by the proceeds from sale of land. They were reporting on an area they had never seen, but used Singapore as a guide. They were doubtful about the success of any scheme of agriculture, but thought a "commercial emporium" might be developed in spite of the opposition of the Dutch. They suggested a scheme of town planning, with land prices up to £100 an acre, but were insistent that it would be necessary first to spend £25,000 on developing the area, and to send out a force of surveyors, sappers, artificers and labourers to carry out the necessary work. Russell declined to spend any money, but offered to send one officer and 10 men to help in constructive work; they seem never to have arrived. Russell's attitude was to do nothing until he received an authoritative opinion as to the advisability of the retention or abandonment of the settlement. Sir Everard Home, visiting the area in H.M.S. *North Star* in 1843, admitted that it did not look impressive, but he strongly recommended its retention. He stressed the additional security afforded to British shipping in the adjacent waters. He had great hopes for future trade in the Archipelago, and thought the conclusion of the war in China[1] would be followed by important developments in trade between China and Australia, particularly in wool. After receiving this report, the Government decided that they "would not be justified in incurring any greater expense than is already allowed for the maintenance of the settlement, nor in planning it on any more regular or permanent footing than at present". The opinion of most subsequent visitors was in accord with that of J. B. Jukes, naturalist of H.M.S. *Fly*, who wrote (1847):

> I believe it to be utterly worthless as a colony, or as an agricultural or commercial possession, and consider the only argument for our continuing to hold it must be a political one.

As the high hopes for the establishment gradually faded, it became more and more a remote neglected outpost. The naval vessel attached was withdrawn, and means of communication became less frequent. Even in 1842, when Bremer was pressing for a monthly service from Sydney during the favourable season, Macarthur was complaining that there had not been a ship from Sydney for 14 months. For a time a vessel from Singapore visited the port once annually, but that ceased. It was visited by occasional men of War—*Beagle*, *Bramble*, *Fly* and *Rattlesnake*—on surveying and other duties, but they rarely had any excess stores. The garrison was frequently short of footwear, and occasionally of clothes. H.M.S. *Alligator*, now a store ship, accompanied Everard Home to the settlement in 1843, and brought surplus provisions remaining after the war in China, but much of it was of poor quality. Indeed, the food generally was below standard. John MacGillivray, F.R.G.S. (1852), who lived four months in the settlement wrote:

> There is probably no vessel in H.M. Navy, no matter where serving, the men of which are not better supplied than were the residents of Port Essington. In the matter of food the garrison has always been poorly supplied. I have seen them obliged to eat bread [biscuit] not fit for human food, the refuse of stock on hand after the war in China.

FIGURE II.

Victoria Square, Port Essington, in 1846. (From "Narrative of the Surveying Voyage of H.M.S. *Fly*".)

Yet there was none other to be had. There were limited periods when it was possible to give some supplies to visiting ships, but, in general, sufficient vegetables for even the garrison were available for only two or three months of the year. However, in both 1847 and 1848, Macarthur reported that for some time there was a superabundance of both fruit and vegetables. Cattle were imported, but losses in transit were great; many of them strayed, and others perished from eating poisonous shrubs. In any case, it was uneconomic to kill cattle or buffaloes for food unless they had a visiting vessel as, in the prevailing heat, deterioration of fresh meat was rapid. "So", wrote T. H. Huxley in his "Diary", "the oxen live and the men die."

Agriculture.

From the first, Bremer and Macarthur were most optimistic about their gardening and prospects of agriculture. Citrus trees, bananas, custard apples and coconuts flourished, pumpkins and melons gave much promise, sugarcane was doing well, cotton was promising, and they thought they could grow rice (Bremer, 1839). When a hurricane in November, 1839, demolished most of the first communal garden, a new garden was established on what

[1] The first war with China, terminated by the treaty of Nanking in 1842.

seemed a better site. But the fertile areas were few and limited in extent. The soil was mainly sandy with ironstone detritus; even grass grew sparsely and trees were rather stunted. To unprejudiced visitors it was apparent that in the absence of vast areas of fertile land, and an abundant population, the value of the settlement for agricultural purposes was negligible. Faced with these conflicting opinions, the Colonial Land and Emigration Commissioners sought the opinion of J. Crawford, an expert with several years of administrative experience in Singapore. His report (1843) should have been convincing. There were, he wrote, only a few fertile patches; the remainder was quite unfit for agriculture; the heat at times was excessive, the rainfall was irregular and uncertain, and there were no rivers. It would be difficult to suggestion by W. E. Gladstone (1846) that it might be utilized as a penal settlement.

Trade with the Malays.

The projected "commercial emporium" did not develop. When no merchants from London or Sydney showed any interest in the settlement, efforts were made to persuade traders from Singapore to establish a depot there, but none could be induced to take the risk. The Government was not prepared to spend money in developing the area until it showed indications of permanence, which was impossible unless funds were made available. Bremer was enthusiastic about the prospects, and his optimism was shared by G. W. Earl, who was attached to the settlement as linguist and interpreter, and who, after a tour round the

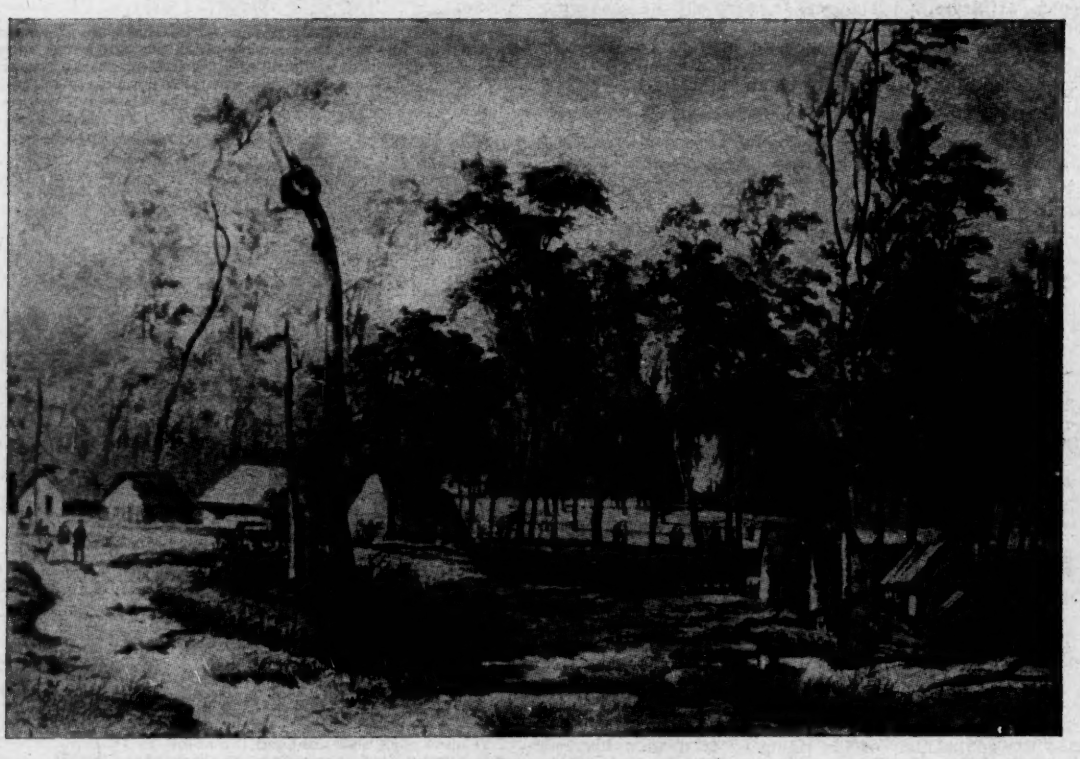

FIGURE III.

Port Essington in 1839. (From a sketch by Mons. Coupil, in *"Voyage au Pole Sud et dans L'Oceanie sur les Corvettes ... L'Astrolabe et La Zelee, 1837-1840"*, Paris, 1846.)

conceive conditions less suitable for tropical agriculture, particularly as the essential cheap labour was not available. It lay outside the direct high road of commerce and while there might be room for an emporium apart from Singapore, it could not be at Port Essington. In contradistinction:

> Singapore is the model and example of an emporium and port of refuge. It is on the high road of all countries lying west and east of it, of some at all times, and of others very frequently. It is accessible at all Monsoons, Ships in their direct voyage actually pass through its harbour, and in wood, water, refreshments, and commercial information, they are well paid for a few hours' detention.

The Commissioners commented that some other authorities were not in agreement with Crawford. They consulted with Sir George Gipps (Governor of New South Wales), who fought throughout very vigorously for the retention of the settlement, and agreed with him that the place ought not to be abandoned, although they rejected a Archipelago in *Britomart*, wrote a comprehensive report on trade conditions. The Malays certainly continued to visit Port Essington—sometimes 500 at a time—and they welcomed Macarthur's protection against the natives, who were still hostile; but their main interest was in catching and curing trepang, and with improved methods their profits increased considerably. Their crews were large, they travelled far, and had little surplus space for trade goods, bringing with them rice, sugar, baskets and mats in limited quantities. While the rice was a welcome addition to the garrison's diet, 50 men on meagre pay could not do much trading, and anything acquired in barter was liable to seizure by Dutch authorities when they returned home. There could be no progress without a larger population.

Efforts to induce the Malays to settle in the area were unsuccessful. Macarthur thought they might come if they were offered liberal terms, including transport. However, they were disinclined to migrate in small numbers. A man of influence among them was necessary to lead them into the area, and to continue to control them and to administer

their affairs—an indulgence permitted to them by the Dutch. But no such man was forthcoming. The great impediment to commerce was the Dutch policy of complete obstruction to British trade with any of their eastern possessions. It might have been practicable to establish trade with independent islands, and at times to evade the Dutch customs, but no one was prepared to take the risk; nor could any of the optimists suggest how Dutch restrictions on trade were to be overcome.

The Haven for Shipwrecks.

The suitability of Port Essington as a haven for succour of shipwrecked personnel was severely criticized almost from its establishment. Admittedly the harbour itself was excellent, but the approach to it was guarded by dangerous shoals,[1] it was low-lying and its entrance was difficult to

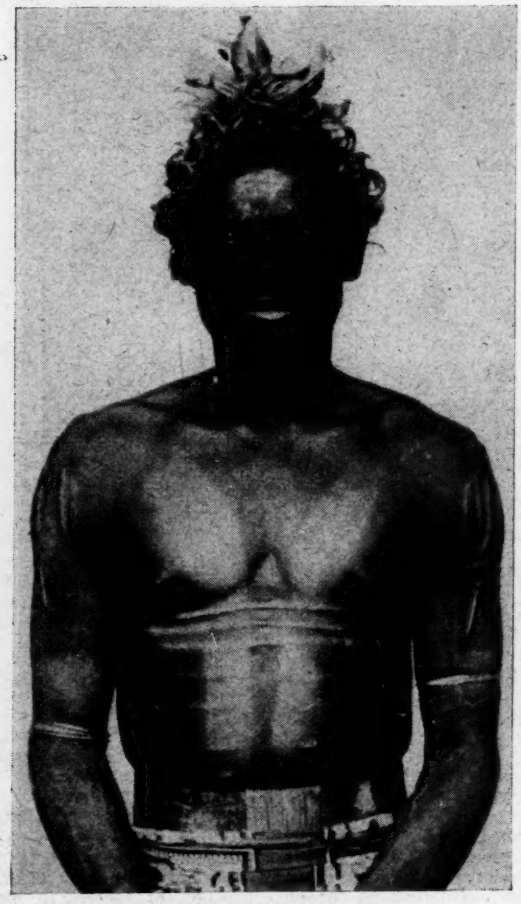

FIGURE IV.
Man—Port Essington tribe.

distinguish from others adjacent. Also it was 600 miles from the reefs where most of the wrecks occurred, and the settlement was 16 miles from the entrance. It was argued that the Cape York area would be much more suitable and much easier and cheaper to supply, although a larger garrison might be needed because of the ferocity of the natives. However, if Port Essington did not meet all requirements, it did provide at least a partial solution to the problem, as, in its absence, shipwrecked personnel must of necessity have sailed several hundred miles further west to Koepang. In fact, Port Essington did occasionally, if rarely, prove very useful. In 1841 the crew of the *Montreal*, which had been wrecked in Torres Straits, arrived in two of the ship's boats: and in June, 1843, survivors of the wrecks of *Hyderabad* and *Couringa* packet, 70 in all, came to the settlement, much to the embarrassment of Macarthur, but fortunately H.M.S. *Fly* was available to take them to Singapore.

The Overland Route.

Increased prosperity in New South Wales in the late 1830's and the growth of trade with India, raised the question of an overland route, so that cattle, horses and goods, generally sent to India and even to Britain, might avoid the tedious and hazardous passage through Torres Straits. Sir George Gipps referred to it in a dispatch in 1840, and as Port Essington was the only northern settlement, it was suggested that it be the terminus of the road. Charles Sturt was interested in the scheme, and E. J. Eyre drew up an elaborate plan involving the use of a coastal ship. Dr. Charles Nicholson moved the appointment of a Select Committee in 1842 to discuss the practicability of such a route, and when its report was presented, Gipps referred it to London with the comment that the direct route proposed offered slender hopes of success, and was unlikely to open up a communication of practicability. He admitted, however, that Sir Thomas Mitchell, the Surveyor-General, not only thought the project practicable, but claimed the honour of leading the party. He also wrote in his dispatch (October 24, 1844):

> I have to inform your Lordship that a small private expedition led by a gentleman called Leichtardt [*sic*] is now on the point of starting out from Moreton Bay to Port Essington direct. Dr. Leichtardt is, I believe, a physician, a German by birth, and a man of considerable scientific attainments.

Eyre thought the expedition would cost £5000; the Select Committee wanted a grant of at least £1000. Leichhardt received no official aid, but raised money, horses and stores by personal canvass of his friends (both his equipment and his stores were entirely inadequate for the task), and left Sydney for Moreton Bay on August 13, 1844. Sixteen months later, on December 17, 1845, seven men headed by Ludwig Leichhardt rode into the settlement at Port Essington, to the amazement of Macarthur and his garrison. They were ragged and hungry, but tremendously relieved to have completed their journey of 3000 miles, and quite emotional at the warmth of their reception. Amongst other hazards, they had been attacked by blacks on June 28, 1845. John Gilbert,[1] collector for the ornithologist Gould, and the most important member of the party, was killed, and two others—Calvert and Roper—were seriously wounded, but they survived. Leichhardt was compelled to relinquish his large botanical collection because of lack of transport, when some of his horses were drowned. Macarthur outfitted them as well as his meagre stores permitted, and after a month's rest in the settlement they returned to Sydney in the *Heroine*, arriving there on March 25, 1846.

The Natives

The natives in the settlement were of the same type as those of Raffles Bay: limbs straight, bodies erect, heads well-shaped and features generally good. They were intelligent, and quite apt in learning anything they deemed useful. They were very prone to steal, particularly food or anything metallic, but it was found much easier to discipline them than was usual in other areas. Most of the credit for this was given to Captain Barker, who, during his command at Raffles Bay, had established great personal prestige amongst the natives, and had brought them fully under control.

[1] The hazards of the port were exemplified in April, 1842, when the vessel *Lord Auckland*, visiting the settlement on her way to New Zealand, ran aground on a shoal. However, she was only slightly damaged, was refloated, and by the combined efforts of garrison and crew was rendered seaworthy and continued on her way.

[1] Gilbert had spent some time in Port Essington. He sailed from Sydney on June 15, 1840, by *Gilmore*, a relief ship sent to the settlement when news was received of the great hurricane of the previous November. He remained there until March, 1841, when he sailed in *Pelorus* to Singapore. In the interim, in spite of intense heat and damage by ants, he acquired a very interesting and beautiful collection of birds, as well as plants, insects, and a few fishes and mammals.

Macarthur was kindly disposed to them, and anxious to improve their living conditions. In September, 1841, he wrote to Sir Gordon Bremer, now the Commander-in-Chief on the India Station:

> The natives suffered severely during the last season from inflammation of the chest and ophthalmia. These poor creatures bury themselves in sand near fires on the cold nights and we observe that in inflammatory diseases of the bowels and chest they use warm sand baths and scald with fire externally. The children have been very much affected in this manner.

He was sorry to see that they had been ensnared into habits of smoking tobacco, as he considered that this induced, at last, a love of ardent spirits, although there was little danger of this at present. On one point Macarthur was adamant—under no circumstances would he allow who surprised the natives by the facility with which he acquired their language, in which he became quite fluent. He had difficulty in maintaining contact with them because of their nomadic habits, and he wished to go far afield to the Alligator River, but Macarthur persuaded him in the first instance, at least, to limit his approach to those in close contact with the settlement, and built a hut for him about ten miles down the bay, to which his supplies were sent every month. He assisted in providing medical attention to the natives. In 1847 there was a severe epidemic of influenza among them, attended by a high mortality—60 to 70 in one small settlement. The medical officer and the Vicar-General worked unceasingly, giving the patients appropriate medical treatment and diet. "Nothing but our assistance" wrote Macarthur, "saved them from an exterminating mortality." They were

Figure V.
Hospital at Victoria, Port Essington, with natives.

Malays to bring into the settlement their "pernicious" arrack, lest they corrupt either the garrison or, particularly, the natives. He suggested that the natives be given an issue of clothing, as had happened elsewhere, as he considered this would conciliate them, and he could obtain equivalent labour in return. His general attitude towards them was one of kindly tolerance; he asserted that, on better acquaintance, they were found to be capable of sympathy and affection to their fellows, and of most humane tenderness in their domestic life.

When the *Heroine* was returning to Port Essington in April, 1846, after taking Leichhardt to Sydney, she ran on a reef, and sank immediately. Included amongst the passengers were three Roman Catholic missionaries. Two of these (catechists), Nicholas Hogan and James Fagan, were drowned, but the third, a Tyrolean, Father Angelo Confalonieri, who had been appointed Vicar-General of Port Essington, managed to survive with the assistance of a large dog, and was brought to Port Essington by a vessel accompanying the *Heroine*. Macarthur equipped him as best he could. He was a kindly man of great culture, ungrateful enough to steal from the Vicar-General, in his absence, all his food and everything else of value he possessed. He found them very difficult to deal with, and considered that the adults were "beyond the barrier of all hopes". He thought the only prospect of success in his mission lay in segregating the children from the adults, so that they might be brought up in ignorance of their parents' "modes and habits of life": but that, of course, the natives would never permit. Don Angelo Confalonieri died of fever on June 9, 1848, and was buried at Port Essington. He was greatly esteemed by the garrison.

In the final days of the settlement, the natives were more difficult to control. When Macarthur tried to discipline them for the murder of two natives of another tribe, they replied insolently that, whilst they recognized his authority in all their contacts with white men, they would deal with other blacks in their own traditional manner.

Health of the Garrison.

In a letter dated September, 1841, Macarthur, for the first time, expressed concern about the health of the troops.

After the cessation of the wet season in the middle of April, there had been two cases of intermittent fever, and also a few of diarrhœa and scurvy—several of them were resistant to treatment, and severe relapses occurred from which recovery was slow (medical stores were deficient). He had established a convalescent camp half-way up the harbour where it was much cooler, and the change had hastened their convalescence. The most common disorder was a persistent ophthalmia—due, he thought, to the strong prevailing winds—but there was also much minor bowel disturbance (this may have been due to the water from some of the wells). He encouraged his troops to play cricket and other sports to keep them fit. He had recently invited the crews of *Britomart* and *Beagle* ashore "to enjoy the recreation that rural sports afford", and was immensely pleased when all the prizes were won by the garrison. Early in 1842 they had abundant food, and the general health had greatly improved. "We have had no sickness except from the same parties who have been complaining from the first." Assistant Surgeon Frederick Whipple furnished a report on the general health of the community for three months following July 1, 1842. The only disquieting feature was the occurrence of five cases of intermittent fever, but these were mild and yielded readily to treatment. Most of the other patients suffered from minor ailments, although there were two cases of phthisis and two of syphilitic ulceration. Seeing that the detachment had been living for upwards of four years exposed to the enervating influence of a tropical climate which was so frequently fatal to European settlers, he thought it necessary to state the reasons why, in his opinion, the general health had been so good. These were (*a*) the favourable site of the colony, exposed to the full benefit of the sea breeze, the dryness of the atmosphere and absence of swampy grounds (many visitors to the settlement did not agree with him); (*b*) the regular and temperate mode of life; (*c*) abundant vegetables. But his optimism was not justified. Early in 1843, a malignant form of malaria, introduced into the settlement by a carrier from a visiting ship or proa, profoundly affected the health of the garrison. Visitors remarked how ill and debilitated the troops appeared, and when the garrison was relieved in November, 1844, more than half of them had died or been already invalided home. Whipple had left the settlement in August, 1843, exchanging to H.M.S. *Fly*, replacing Assistant Surgeon Sibbald, who returned to Sydney with the relieved garrison; but he was asked to explain the reason for such a marked deterioration in the health of the garrison. In his reply, dated March 23, 1844, he wrote that, from the initiation of the settlement until the beginning of the dry season in 1843, there had been only one death there from illness—that of a sailor from H.M. brig *Pelorus*, who died of dysentery. However, in 1843, there were numerous cases of remittent and intermittent fever, with several deaths, four from the settlement, and three from the brig *Carnelion*. All the deaths occurred within five to eight days from the onset of symptoms. Of 40 men in the garrison, only two were unaffected. He left at the settlement 18 patients, though many of them were convalescent. The whole party were very debilitated, not only from malaria, but also from prolonged exposure to a tropical climate, and he considered that their relief was a matter of urgency. The cause of this serious epidemic, he thought, was excessive rainfall.

> The immense body of water collected in the numerous lagoons contiguous to the Settlement—the strong evaporation of which, and the decay of vegetable matter abounding in these situations causes considerable malaria to exist.

He thought that the general health improved considerably when there was available a good supply of animal food and beer, but this rarely happened.

The garrison was relieved on November 19, 1844. Macarthur remained as Commandant, and the new doctor was Assistant Surgeon Tilston. Lieutenant Lambrick, the Quartermaster, was so ill informed as to conditions prevailing in the settlement, that he brought with him his newly-wed wife—with disastrous results; three of the other ranks were also accompanied by their wives. But the malaria still took its toll. When H.M.S. *Fly* visited the settlement in June, 1845, all the new arrivals had had fever, they were sallow and emaciated and already four had died. Macarthur did not think the relieving troops had the physical stamina of their predecessors. In the settlement there was no longer any talk of extensive tropical agriculture or of a commercial emporium. In view of their ill-health and the heat, they were usually able to work only about four hours a day, and these were devoted to repairing the ravages of the hurricanes and the termites, which seemed to be winning all along the line. In May, 1846, Macarthur wrote to the Colonial Secretary asking for an additional medical officer. He had had a bad day. First, Lieutenant Wright, one of his officers, had had an epileptic seizure, and it was necessary to begin the prolonged arrangements for his relief. Next, Assistant Surgeon Tilston saw him to report a death from fever. He then, with great emotion, said that he was at the end of his physical and mental resources, and could no longer carry on—in fact, he thought he was likely to die. He asked that an urgent application be made to the Lords of the Admiralty for an additional medical officer, preferably one with some knowledge of tropical diseases, for, he added, "the fever here differs from any that I have seen described". Apparently, in addition to his other worries, Tilston had also received some distressing news from his family. Macarthur was most sympathetic, realizing that Tilston had no time off throughout the whole 24 hours, and that his position was much worse now that there was no longer a small warship with its own medical officer located in the port. With Macarthur's kindly handling, Tilston improved and was able to carry on, which was just as well, for Macarthur's letter of May 21 was not received in Sydney until December 7. A report from Dr. William Dawson, the Deputy Inspector-General of Hospitals in Sydney, three weeks later, agreed that a garrison in the tropics should have an experienced doctor, and that, although to secure such a man for a temporary duty would have necessitated a high rate of pay, he would have recommended it to His Excellency, only that a similar application had been made to the Adjutant-General of Marines, and, in view of the lapse of time, a doctor would reach the settlement from London much sooner than one could be made available from Sydney—a view in which the Colonial Secretary concurred. This was not very helpful. However, some time in 1847 Surgeon Crawford arrived in the settlement, to take over medical control and supervise Tilston, who was still on duty.

In October, 1847, Macarthur wrote: "The establishment is now enjoying as good health as I shall ever expect to see, but four men have been invalided whose lives appear to depend on change of climate". In the next year there was constantly a large sick list—quite often no very active disease and only a few patients in hospital, but a general state of nervousness and debility throughout the community, and an intense desire to get away from the place. Surgeon Crawford, however, had several severe bouts of fever. Early in 1849 came the worst epidemic of all. In February, Surgeon Crawford became once more seriously ill, with high temperature and delirium, and under the extra stress imposed on him Tilston caved in completely.

> On 25th February I considered Dr. Crawford to be in a very critical state whilst I was really alarmed for Mr. Tilston whose illness I discovered had been complicated with nervous disease for sometime past. On this day I felt the first symptoms of fever myself and a serious sickness ensued accompanied by distressing palpitations. Lieut. Dunbar also laid by—severe fever probably—but he checked it by strong doses of mercury. Now Captain Lambrick alone of all the Officers was on his feet and by a good providence maintained his health throughout. From this period the fever constantly fixed its fangs upon us until every man (I think there is no exception) had been laid up. In April we numbered 23 patients exclusive of the Officers and could furnish only 3 men for night watch. On March 4th Mr. Tilston's suffering ended. His disorder was so complicated that it was fearful to do anything for him but what was obviously needed. On the 10th another private died—before the fever attacked him he had suffered so much as to be nominated for invaliding. On the 24th another private died. Dr. Crawford at this time was much recovered and enabled to prescribe. He tells me this was a case of English cholera. There had been something of the same

nature among the natives. (Macarthur to Colonial Secretary, 1849.)

All this sickness interfered greatly with the work of the settlement, and destruction by the termites increased apace. Even the hospital—the best building—was in a bad state; when it rained beds must be moved, and the surgeon had to put a tent-like canopy over his own bed; everyone was unhappy and awaiting relief; all of them had had repeated attacks of intermittent fever and "another fever of a more deadly character occasionally appeared and, operating on a previously debilitated constitution, frequently proved fatal" (MacGillivray, 1852).

Macarthur had now been 11 years at Port Essington, throughout which he had worked hard, with scanty official encouragement and frequent embarrassment from visiting naval officers, to ensure the success of what he regarded as a great and worthwhile experiment. His habits were simple and austere, but apparently his efforts to maintain morale in a dispirited and enervated community were not always appreciated. The biologist, T. H. Huxley, visiting the settlement in H.M.S. *Rattlesnake*, wrote in his usual forthright style:

> As for the place itself, it deserves all the abuse that has ever been heaped on it. It is fit neither for man nor beast: to speak of it as a settlement is a mere abuse of words—the country in the neighbourhood is the most wretched, the climate the most unhealthy, the human beings the most uncomfortable, and the houses in a condition the most decayed and rotten.

He admitted that the officers were united in extreme kindness and hospitality to their guests, and that pineapples were available in abundance, but "the respected Captain Macarthur is, with all reverence, one of the most pragmatical old fogeys I ever met with and contrives to keep the people in his command continually in hot water". This criticism was very unfair to Macarthur. The British Government, having established the garrison at Port Essington, had dealt with it in a very niggardly manner, and when it was realized that the objects for which it was founded were incapable of achievement, the settlement had been grossly neglected. The garrison was, in the main, left to fend for itself with inadequate communication, frequently lacking food, clothing and even medical stores. From the small party, further lessened by sickness, discouraged by the monotony of their existence and worried by the uncertainty of their future, little more could be expected than that they should be employed in rendering their own condition more tolerable. A much fairer and more sympathetic assessment of him had been made by Owen Stanley some years previously:

> Captain McArthur appears to have done all in his power with the limited means afforded by a population of only 55 souls. Interfered with and opposed as he has hitherto been by the Commanding Officers of Men of War touching at the port I only wonder he had temper to persevere in such uphill work. The 'Pelorus' even took away his only brickmaker.

Macarthur was invalided shortly after his return to Sydney.

The Final Stage.

In April, 1849, Earl Grey wrote to the Admiralty, pointing out that the settlement was costing £3204 *per annum* without any commensurate advantage.

It seemed that none of the objects for which it was founded could be fulfilled. A company, formed to supply a steamer service between Britain and Sydney via Torres Straits, would form a coaling station at Cape York, but did not propose to call at Port Essington unless subsidized for doing so. Accordingly Grey did not consider that there were "sufficient motives" for continuing the establishment at a cost out of proportion to the benefits derived, and he asked for advice on the most convenient way of moving the garrison from Port Essington. At the same time he desired to hear their views on the expediency of forming a settlement at Cape York.

It is rather surprising that, in the ensuing discussions, the general ill-health prevailing at Port Essington was barely mentioned; only in one instance is reference made to the fact that it was "at times unhealthy" (rather an understatement, surely).

There was almost unanimity among the people consulted as to the superiority of Albany Island near Cape York for the purposes required. The only dissident voice was that of Sir Gordon Bremer, who, admitting his imperfect knowledge of the Cape York area, said he was not impressed by it, and stressed the ferocity of natives of the Torres Straits. He still argued that if suitable facilities were provided at Port Essington, "a mercantile adventure of a most profitable nature should follow".

Grey's decision was that immediate measures should be taken to close the establishment at Port Essington, but he was not at present prepared to recommend incurring the expense of maintaining a settlement at Cape York which, he admitted, future circumstances might render necessary.

On November 12, 1849, the Honourable K. Keppel arrived in H.M.S. *Menander* to carry out the evacuation. All buildings were destroyed in order to remove any inducement to other parties to settle, and because it was thought that if they remained they might provide a cause for bloodshed between local natives and other tribes. Keppel thought that a few members of the garrison who were interested in shooting were a striking physical contrast to their fellows, and concluded that the ill-health of the community was as much due to lack of suitable occupation, physical and mental, as to climatic conditions and deficient diet. A few days after the arrival of the *Menander*, her surgeon, John Clarke, died—"a man who by his kind and gentle manner and his amiable disposition had endeared himself to us all". Clarke had acquired some infection at Hong Kong from which he never completely recovered. He was interred in the cemetery beside Mrs. Lambrick and her daughter, who had perished some months previously. On November 30, 1849, the garrison, headed by Macarthur, and preceded by the band of H.M.S. *Menander*, marched with all due ceremony out of the settlement which had so many unpleasant memories. But these proceedings "did not excite sufficient interest among the natives (I must except a few of the softer sex) from their search for what they could find amongst the ruins of the buildings" (Keppel, 1853). The next day the *Menander* sailed for Sydney.

After the relief party had arrived in the settlement on November 19, 1844, the garrison, including Macarthur, numbered six officers and 58 men. In the ensuing five years, they fared as shown in Table I.

Table I.

Fate of Garrison.	Officers.	Men.
Died	1	12
Invalided	1	13
Evacuated ..	4	33
Total	6	58

Why was the garrison kept at Port Essington so long after it was obvious that the settlement was a failure? The reasons seem to be entirely political. The Colonial Land and Emigration Commissioners had earlier (1840) written to Lord John Russell:

> Apart from its importance as a naval station or as a commercial depot, it is in the highest degree desirable that the Northern Coast of New Holland should be made British territory by actual occupation, and its importance in giving command of navigation in Torres Straits and of the commerce of the Indian Archipelago might possibly have seemed an object worthy of the attention of the British Government even at some cost to the country.

And in the final discussion, Admiral Beaufort, Naval Hydrographer, wrote:

> The Peninsula or Port Essington might perhaps be chosen for a penal settlement, or if not, it might well be left until the tide of immigrants should spread in that direction were it not for the danger that some other nation, French, American, or Dutch from the neighbouring Islands might take possession of a coast

which we should then have for the second time abandoned—the consequences of which might cost us more than keeping up a dozen useless establishments.

Acknowledgements.

To Professor E. Ford, who spent some time in the area in the late thirties, I am indebted for much useful advice and for the photographs of Melville Island. The illustrations of the Port Essington Settlement are reproduced by kind permission of the Trustees of the Mitchell Library, Sydney. The sources from which this account is derived are all found in that library, and I gratefully acknowledge the skilled assistance of the library staff.

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PLAY THERAPY[1]

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WITH the subject of play therapy are associated some confusion about definition and doubt of its value.

We can define play therapy as the use of play and particular play techniques in the psychiatric treatment of children. The doubt of its value arises when play therapy is regarded as a complete treatment for the emotional disturbances of children, when it should be considered only as a possible part of the treatment of the disturbance.

The history of its development dates from the work of Helga von Hug Helmuth in Germany in the early part of this century. She was one of the first psychiatrists to describe and to understand the behaviour of children. She used drawings and play materials to help her in the study of the child, but she did not create any specific play techniques.

Play techniques were first used by Mélanie Klein in Berlin from 1919 and in London from 1926, and by Anna Freud, working in Vienna from 1920 and in London from 1938. David Levy and Louise Despert were working along similar lines in New York from the 1920's. The influence of these four child analysts has been widespread.

The Use of Play Materials in the Treatment of the Emotionally Disturbed Child.

With the child of 10 to 12 years and younger, play is a useful adjunct to the direct discussion of his problem with him. To quote Lauretta Bender: "The play situation can be therapeutic if it tends to offer the child opportunities for articulateness." Without play materials, one can find the interview developing into a question and answer situation. While playing, the child talks more freely, and through the play one can obtain material for further discussion. Drawings are particularly useful in this way.

Play also helps to anchor the over-active child. This type of child soon becomes restless in an interview if one relies upon discussion alone. The introduction of play materials allows one to prolong the discussion.

For short-term psychotherapy, paints or coloured pencils and paper, or blackboard and chalks, *plus* a few doll figures, may be all the play materials that are necessary. For the more disturbed young child who requires a longer period of treatment, one will need a greater variety of equipment, e.g. sand tray, water, clay, dolls and doll furniture, cars, boats, trains, toy figures, animals, guns and building material.

The question of whether or not to use a play room depends upon the type of consulting room which one has. The room which is furnished as an office. with telephone, cupboard or filing cabinet and desk with many drawers, is unsuitable as a room for interviewing over-active children. It is not sufficient to say to the child that one does not touch the telephone or look into drawers or cupboards, for he still attempts to do so. It is better to see the child in a room where there are fewer distractions.

Play material helps in the establishment of rapport with the child, but the therapist must not rely too much upon play materials. It is of primary importance to develop one's ability to form a positive relationship with the child through discussion, and to use aids only secondarily. Play therapy has developed to its present extent, to some degree because many therapists have not had the necessary experience and understanding to have been able to talk with children simply and in a way which is acceptable to them. In the words of Lauretta Bender: "Play is a useful prop for those working with children who are not comfortable in their relationship with children."

It is useful to observe the child at play. The so-called normal child uses play materials constructively and is able to play imaginatively. The disturbed child will show special features in his play, and by observing him, one can confirm and add to information given to us by his parent. The child may avoid a particular type of play, or repeat one pattern of play, or fail to concentrate on any play material for long. It is usually necessary to observe him over several sessions before one can make reliable judgements from his play alone. However, one does not spend a number of sessions in merely observing the child at play.

Play materials are used educationally in child-guidance work. Occasionally, through ignorance or poverty of the parents, a child is quite unfamiliar with play materials, and has to be introduced to toys and equipment suited to his age group. The disturbed child who is already familiar with play materials can be educated to develop his skills, and to play more imaginatively, and thereby to gain more satisfaction from his play.

Through play in the presence of an accepting adult, the child is said to be able to act out situations and to relieve tensions more readily than in ordinary play. This may be so, but tensions will quickly build up again unless one is primarily concerned with improving the child's relationships.

Group play under good supervision helps all types of emotionally disturbed children. The group must be kept small if it is comprised of disturbed children only, and one must exclude a very aggressive child if that child repeatedly disrupts

[1] Read at a meeting of the Australasian Association of Psychiatrists, Brisbane, 1956.

the group. The kindergarten teacher or group social worker would be an asset to any child-guidance centre, to take the waiting children in a group or to have selected groups of children for play. Unfortunately, it is rare to have a trained group worker attached to a clinic. One or more of the team members may conduct a play group within the clinic, but the training of the team members has usually been with individuals rather than with groups. One does therefore rely on outside groups to a large extent, though one hesitates to refer a child who is seriously disturbed.

Play Techniques.

Interpretation to the Child of His Play.

The analyst believes that the child's fantasies, anxieties or defences, expressed in his play, can be directly interpreted to him with the result that further material comes up in his play. This approach is said to correspond with the use of free association in adults. Direct interpretation of their play to children is used to a varying degree by different analysts. Anna Freud uses interpretation only sparingly, and emphasizes the importance of educational, as well as analytical, measures. She stresses also the need to keep in touch with current happenings in the child's environment through work with the parents.

In therapy with the child, many of us prefer to use what the child tells us, rather than what his play appears to reveal to us. We try to treat the child in the same way as we treat the adult, but at the child's level. We use, to guide the discussion, all we know about the child from previous discussions with him and with his parents. We can use material suggested to us by his play, but his play is mainly meaningful to us because of what we already know about him, and it may not contribute very much extra knowledge. Asking the child to tell us about his play and his drawings or paintings, does help to bring the child into the psychotherapeutic situation. When the child tells us something, he is usually ready to discuss it further, whereas he may not be ready to accept direct interpretation of his play at a particular time, even though the interpretation is the correct one.

Structured Play Situations.

This technique was first employed by David Levy, and is still used by some of his followers in what they call release therapy. This particular play technique uses play situations dealing with various relationships—for example, the arrival of a new baby in the family, punishment by parents, illness or hospitalization.

Doll figures are used to depict the situations, and the child is encouraged to act out the experience which appeared to have precipitated some of the symptoms. Those who use this technique claim that the release of tension occurring in the acting out of the situation is therapeutic. However, most of the children who come under treatment have long-standing problems in family relationships, and the so-called traumatic events are usually not as significant as disturbed parent-child relationships persisting from early infancy.

Control of Aggression in the Playroom.

A play session should never be allowed to degenerate into an uncontrolled show of aggression and destruction by the child, and limits have to be set therefore. The over-active, aggressive child will test our patience, and it is important to maintain control of the situation, and to conclude the play session if the child is overstepping the limits in spite of due warning. The play material is often attacked vigorously, and this must be permitted to some degree, but not to the extent of allowing the throwing of toys, water, sand, clay or paint about the room. The child is not being helped to conform socially if there are no restrictions on this type of behaviour at a clinic, under the misapprehension that it is a therapeutic release of aggression.

Associated with the control of the child in the play room is the need to expect the child to appreciate good play materials, and to help in the putting away of materials at the end of a play session.

Those who are not familiar with a kindergarten set-up, and who use a playroom, are too often content to have broken and shabby equipment, and to leave toys and material scattered about the room at the end of a play session. This is scarcely educational or therapeutic for the child.

In a clinic, several people may use one playroom, and if no one is responsible for the care and the proper use of the equipment, the playroom becomes a hindrance rather than a help in the treatment of the child.

Regressive Behaviour in the Playroom.

This is not uncommonly seen and is encouraged by some therapists. If the child wants to play at being a baby for a time, it is reasonable; but I feel that the best result will be achieved if, on the whole, one encourages the child to interest himself in play at a level more appropriate to his age. If he cannot do this, we may have to be content to build up gradually from a lower level, but not necessarily from the infancy level.

Conclusion.

Play therapy, which is an indirect approach to the child, should not be used to the exclusion of direct psychotherapy, for any child who is able to talk can enter into some sort of discussion relating to himself. One can become completely lost from the child in the play set-up unless the play is used purposefully and as an aid to direct discussion.

In addition, therapy with the parents should always accompany therapy with the child, and at least as much time should be spent with the parents as with the child.

I think that the most effective way of working in an out-patient setting is for one person, who is usually the psychiatrist, to work with the parents and with the child. If the therapist is not a psychiatrist, he or she, whether a psychologist, social worker or play therapist, should be well trained to deal with children and parents, and the problem must be one which the therapist is capable of handling.

In a residential psychiatric centre or hospital for children, with more seriously disturbed children, it is often impossible for the psychiatrist alone to do the individual work that is necessary with the child and the parents. Therefore, the psychologist or play therapist may be working with the child individually in play sessions under the direction of the psychiatrist, whilst the psychiatrist also sees the child at regular intervals, using play when necessary. The psychiatrist also works with the parents, using the social worker for some of the interviews with them.

Play therapy with the child, in the clinic or consulting room or in an in-patient setting, then becomes a small but valuable part of the whole treatment of the emotionally disturbed child and his parents.

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NYLON REPLACEMENT OF AORTIC SEGMENTS: AN EXPERIMENTAL STUDY.

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By the turn of the century, arterial suture by various methods had become an established experimental procedure as a result of the work of the early pioneers, and by 1903, 30 clinical cases of lateral suture of arteries had been collected and published (Höpfner, 1903). The circular suture of blood vessels had been widely investigated, and Alexis Carrel in 1902 published his well-known triangulation method which, according to a contemporary (Watts, 1907), "while differing very little from certain methods previously employed, has greatly facilitated such sutures". It is this method which forms the basis of all methods of blood vessel suture used today. Carrel continued this work first in France and later in Chicago. In 1906 he and Guthrie reported experiments showing that segments of autogenous vein could be transplanted into arterial gaps and could

[1] Work done during the tenure of a Research Fellowship from the Retail Traders' Association of New South Wales.

function as a portion of the artery. The prophetic conclusion of their article is that "clinically, the transplantation of veins may have applications".

As time passed, almost all the methods of vessel replacement and preservation that have been resurrected in the past 10 years were tried experimentally. Until 1949, clinical opportunities for vessel grafting were mostly beyond the surgical horizon except in wartime. Even then, the difficulties of surgery in the field and the lack of training of surgeons in methods of vessel suture made it almost impossible to replace damaged arterial segments. Tuffier's silver tube had a limited vogue in the first World War, and allowed a few days for the development of collateral circulation before it sloughed out.

In 1945, Blalock and Taussig published their operation for the tetralogy of Fallot, and four years later Gross and his colleagues gave a demonstration of the need for artery grafts in non-traumatic surgery, when they reported nine cases of tetralogy of Fallot in which Blalock's operation could be performed only by interposing a piece of donor artery betwen the subclavian and pulmonary veins (Gross *et alii*, 1949). The technique was soon being tried for a variety of other conditions. Among these conditions were coarctation of the aorta in those cases (one in six or seven, according to Gross, 1951) in which the gap left after excision of the narrowed and diseased portions was too great to be bridged by end-to-end suture. Aneurysms and occlusions of the abdominal aorta were excised and replaced, with good immediate results (DeBakey and Cooley, 1954; DeBakey *et alii*, 1954). In the smaller arteries such as the popliteal, which was mostly replaced for arteriosclerotic blocks, results were disappointing, but a newer tchnique of by-pass by end-to-side anastomosis, leaving the occluded segment in position, has been more encouraging (Linton, 1955). In the recent Korean War, forward surgeons, trained by intensive practice on animals, achieved success with autogenous vein grafts in the peripheral arteries of the wounded (Jahnke and Seely, 1953). Obviously the young soldier with a traumatic arterial wound is a much better subject for grafting than the arteriosclerotic subjects who provide most of the patients needing such grafts in peacetime. In these ways the need for a bank of material for vessel replacement was created.

Artery banks were estabished in many places, but practical difficulties were encountered, and many banks have never contained more than a few pieces of artery. The main difficulties may be summarized as follows: (i) Donors must be young, and must not have died of infectious disease or cancer. This virtually means that they must have died violent or accidental deaths. After such a death, the body is usually examined by a forensic pathologist and not at a hospital. (ii) Material must be obtained sterile, within a few hours of death, under aseptic conditions. This means an operating theatre for all practical purposes. (iii) Because donor material is so hard to obtain, a method of storage which is satisfactory for a prolonged period is necessary; this requires either a deep freeze, which is simple, or a freeze-dryer, which requires a fair amount of technical facilities and equipment.

It has been shown that donated artery need not be living to obtain a successful result (Deterling *et alii*, 1951). The living tissues soon perish in fresh homografts, but the graft acts as a scaffolding for the ingrowth of fibrous tissue from the host into its wall. In animals a new intima is formed; but in man Creech *et alii* (1956) found the endothelial lining still incomplete after two and a half years, and much of the intima still fibrinoid material.

Knowledge of the fact that the donated artery need only act as a conduit for the blood and as a scaffolding for host tissues has led to the development of two main methods for overcoming the difficulties of obtaining donor material, as follows: (*a*) Sterilization of arteries by storage in weak solutions of antiseptic agents. The object here is to sterilize and preserve the arteries without denaturing the proteins of the elastic tissue on which the integrity of the segment depends. Among substances used have been alcohol (Kimoto *et alii*, 1954), 4% formalin (Peirce *et alii*, 1949) and β-propiolactone (Trafas *et alii*, 1954). (*b*) The use of synthetic cloth (Voorhees *et alii*, 1952).[1] This can be prepared to size and boiled. As with dead donor material, these synthetic materials maintain the blood channel and act as a scaffolding for the ingrowth of host tissues.

It is not proposed to discuss the merits of the various methods outlined above or the various cloths that have been used. Each method has its supporters, and it seems probable that different methods will prove best for different pathological or working conditions.

FIGURE 1.

The field of operation showing the prosthesis in position.

Method and Materials.

The object of the present research was a practical one — namely, to try out a particular cloth for arterial replacement for our own use. The cloth used was kindly given by Dr. Harris B. Schumacker, junior, of the Department of Surgery, Indiana University Medical Center, to Mr. F. F. Rundle, F.R.C.S., who supplied it to us. The cloth is a nylon filter fabric. It is an off-balance taffeta, constructed of multifilament highly twisted yarn strands with a warp count of 232 and a filling count of 98. The yarn is 70 denier high-tenacity nylon with 35 turns per inch length. After being autoclaved, the fabric has such a resistance that a pressure equivalent from four to five inches of water will cause it to pass 20 cubic feet of air per minute through a square foot of material.

Sheep were used as the experimental animals, and the artery chosen for replacement was the thoracic aorta. Sheep are notoriously prone to hind-quarter paralysis after clamping of the aorta, and will not even tolerate clamping of the abdominal aorta for more than 30 minutes. In man the abdominal aorta can be clamped without noticeable ill-effects for hours, provided the clamp is below the renal arteries. Hypothermia was used in the experiments to prevent the hindquarter paralysis.

The animals, when shorn, weighed between 40 and 70 pounds. They were anæsthetized by injection of pentobarbitone ("Abbott's Veterinary Nembutal") through an indwelling polythene catheter into the jugular vein. A thermometer, which could be read at a distance, was inserted into the rectum, and the animal was immersed, except for the head into an iced bath at 5° C. Cooling was rapid, and within 30 minutes the animal's temperature had generally fallen from the normal 39° C. to 32° C., when it was removed from the bath. The temperature would then drift down to about 29° C. Respiration was manually

[1] The word "graft" is avoided here in references to synthetic cloth used to replace a segment of artery, although it is frequently employed in this sense. A graft, however, is defined as "a portion of tissue, such as skin, periosteum, bone, fascia or, rarely, an entire organ, used to replace a defect in the body". ("Blackiston's New Gould Medical Dictionary", 1956).

TABLE I.

The Main Changes Seen About Nylon Prostheses in the Aortas of 17 Sheep.

Time Between Operation and Death.	State of Lumen.	Changes Inside Prosthesis.	Changes Outside Prosthesis.	Changes in Ends of Aorta.			Changes Outside Cut End of Aorta.	Comment.
				Necrosis.	Fibrosis.	Cells.		
4 hours.	Patent.	Fibrin layer about 2 mm. thick.	Fibrin only. Adjacent tissue not attached to prosthesis.	Not visible.	No.	No.	None.	Killed.
2 days.	Patent.	Fibrin layer less than 1 mm. thick.	Fibrin only. Adjacent tissue not attached to prosthesis.	Early changes in small areas.	No.	Polymorphs.	None.	Acute dilatation of the stomach. Animal died.
3 days.	Patent.	Fibrin layer about 2 mm. thick in distal 1 cm. and about 1 mm. thick elsewhere.	Adipose tissue attached by fibrin in patchy fashion.	Small areas.	No.	Polymorphs and macrophages.	None.	Killed.
4 days.	Patent.	Fibrin layer 2 mm. thick over distal 8 mm. and less than 1 mm. elsewhere.	Adipose tissue attached by fibrin, especially over seam of prosthesis.	Small areas.	No.	Polymorphs and macrophages.	Polymorphs. Capillaries and fibroblasts proliferating.	*Empyema thoracis.* Killed.
7 days.	Patent.	Fibrin less than 1 mm. thick. Fibrous tissue grown in up to 1 mm. each end.	Adipose tissue weakly attached in a few places. Vessels and fibroblasts in adipose tissue becoming enlarged and numerous.	Small areas.	Fibroblastic activity in adventitia.	A very few macrophages.	Polymorphs. Capillaries and fibroblasts proliferating.	*Empyema thoracis.* Killed.
10 days.	Patent.	Fibrin layer about 1 mm. thick, varying greatly.	Adipose tissue more firmly attached. Capillaries and fibroblasts prominent.	Larger areas.	Fibroblastic activity in adventitia.	A few macrophages.	Fibrous tissue being laid down.	Killed.
14 days.	Occluded.	Clot almost completely occluding lumen. Fibrous tissue grown in 1 mm. each end.	Capillaries and fibroblasts advancing into fibrin. Adipose tissue attached in some areas.	Extensive.	Fibroblasts in aortic media.	Macrophages and a few polymorphs.	Fibrosis advanced.	Prosthesis almost occluded by clot. Died.
15 days.	Patent.	Fibrin about 1 mm. thick. Fibrous tissue grown in 1 to 3 mm. each end.	Fibrous tissue being laid down. Adipose tissue attached in some areas.	Fairly extensive.	Fibroblasts in aortic media.	Macrophages and a few polymorphs.	Active fibrosis, many macrophages.	Killed.
23 days.	Patent.	Fibrin less than 1 mm. thick. Fibrous tissue grown in 1·5 cm. from one side.	A fairly thick layer of fibrous tissue formed. Adipose tissue attached in some areas.	Fairly extensive.	Fibroblasts in aortic media.	No.	Fibrous tissue maturing. Some macrophages.	Killed.
29 days.	Patent.	Remaining fibrin about 1 mm. thick. Fibrous tissue joined across graft at one point.	Moderate layer of fibrous tissue. Adjoining tissue firmly attached.	Extensive.	Fibroblasts in most necrotic areas.	A very few macrophages and lymphocytes.	Fibrous tissue maturing. Some macrophages.	Killed.
44 days.	Patent.	Very little fibrin left. Fibrous tissue almost completely covers.	Thick layer of fibrous tissue. Adjoining tissue firmly attached.	Moderate amount.	Fibrosis becoming less active.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	Found dead, cause unknown.
51 days.	Patent.	Fibrin about 1 mm. thick. Fibrous tissue grown in 4 mm. from each end.	Mature fibrous tissue. Adjoining tissue firmly attached.	Very little.	Inactive fibrous tissue. Calcification in media.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	A few fibroblasts passed through the nylon from the outside. Killed.
56 days.	Patent.	Fibrin about 1 mm. thick. Fibrous tissue grown in 1 cm. from each end.	Mature fibrous tissue. Adjoining tissue firmly attached.	Moderate amount.	Active fibrosis.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	Killed.
68 days.	Patent.	Fibrin about 3 mm. thick at one area. Fibrous tissue grown in 1 cm. from each end.	Mature fibrous tissue. Adjoining tissue firmly attached.	Little.	Inactive fibrous tissue; calcification in media.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	Small islands of fibrous tissue in the inner fibrin layer ahead of the fibrosis advancing from the aorta. Killed.

TABLE I.—*Continued.*

The Main Changes Seen About Nylon Prostheses in the Aortas of 17 Sheep.—Continued.

Time Between Operation and Death.	State of Lumen.	Changes Inside Prosthesis.	Changes Outside Prosthesis.	Changes in Ends of Aorta.			Changes Outside Cut End of Aorta.	Comment.
				Necrosis.	Fibrosis.	Cells.		
86 days.	Patent.	Fibrin about 2 mm. thick. Fibrous tissue grown in 1 cm. from one side and 4 mm. from other.	Mature fibrous tissue. Adjoining tissue firmly attached.	Little.	Inactive fibrous tissue.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	A few fibroblasts passed through the nylon. Killed.
119 days.	Patent.	Remaining fibrin about 1 mm. thick. Fibrous tissue joined and covers four-fifths of inner surface.	Mature fibrous tissue. Adjoining tissue firmly attached except one small area.	Extensive.	Inactive fibrous tissue; some calcification.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	Killed.
137 days.	Patent.	Remaining fibrin about 1 mm. thick. Fibrous tissue grown in 1·5 cm. from one side and 3 mm. from other.	Mature fibrous tissue. Adjoining tissue firmly attached.	None.	Inactive fibrous tissue.	No.	Fibrous tissue now inactive. A few macrophages and lymphocytes.	Old animal. Killed.

maintained with oxygen by means of an anæsthetic bag connected through a carbon dioxide absorber to the intratracheal tube.

The left side of the chest was then opened by resecting the fourth or fifth rib. After the lung had been mobilized, the junction of the aortic arch and descending aorta was bared of pleura, and the aorta was divided between Crafoord coarctation clamps. As far as possible the adventitia was not removed. A tube of nylon cloth was prepared in advance by being folded in two, having a seam down one side with a fine needle on an ordinary sewing machine, and then boiled in tap water. This nylon tube was interposed between the retracted ends. A length of about four centimetres of tube was generally adequate without kinking.

The technique of suture requires special mention. In two of the early experiments the suture line had to be excised and recommenced because of kinking and bunching. This was caused by disparity in size between the nylon tube and the aorta, or by misjudgement in placing the sutures. It was obvious that the technique had to be even more exact with the nylon tube than in an end-to-end suture of an artery. Number 5/0 "Deknatel" on eyeless needles was used, and the cloth was first secured, seam towards the operator, by two everting mattress sutures exactly halving the circumference of the artery and the nylon tube. Another everting mattress suture was then placed in the exact centre front, and, with slight tension on the appropriate mattress sutures, the line was sewn together with a continuous everting stitch of the Carrel type. The artery and nylon tube were then turned over, and the back was completed in the same manner with the preliminary insertion of a mattress suture in the centre. In other words, the suture line was quadrangulated with equidistant mattress stitches. Suturing was generally finished in 40 minutes and the lower clamp released; the upper clamp was released when oozing had ceased. Bleeding from the suture lines occasionally required an extra stitch. On two occasions oozing through the cloth was brisk, and on four occasions there was none. In the remainder of the experiments it was slight. No explanation for this difference was apparent. Pressure with a swab soon stopped all oozing from the cloth, or from the suture line that did not require a stitch. Figure I shows the prosthesis in position.

The pleura was closed over the nylon tube, and the chest was closed without drainage. The animal was warmed in a bath at 40° C., and usually regained consciousness when its temperature had returned to 34° C. It was then removed from the bath and allowed to warm by its spontaneous shivering efforts.

Results.

The procedure was well tolerated, and there were no immediate deaths. One animal died of a thrombosed prosthesis on the fourteenth day. In the others the channel through the prosthesis remained patent. No case of disruption of the suture line occurred. The 17 animals died or were killed at the times shown in Table I.

From each animal the prosthesis was removed with the tissues attached to its outer surface and with a few centimetres of aorta at each end.

After naked-eye examination, tissue was cut for blocks. Two or more pieces were cut across the anastomosis to include both aorta and fabric. The faces of the paraffin blocks were trimmed and then immersed in a solution composed of equal parts of phenol and chloral hydrate in order to soften the nylon. After seven to 14 days, several sections were cut from each block. The nylon was still difficult to cut, and many sections were rather broken up. The sections from each block were stained with hæmatoxylin and eosin. Selected sections were stained with Verhoeff's elastic stain combined with Van Gieson's stain, and others were stained with Masson's trichrome stain.

Table I sets out the main changes seen in the specimens. The events which occurred after the nylon tubes had been inserted fall into two stages.

As soon as the blood was allowed to flow through the prosthesis, coagulation began in contact with the nylon. A layer of fibrin usually less than two millimetres thick was

formed, trapping some blood cells. A smaller amount of fibrin was deposited on the outer surface, from blood which had passed through the fabric and from serum from adjacent tissue. Some of the cellular tissue in the media of the aorta near the anastomosis died. The consequent changes in appearance were recognized at the end of two days. The elastic tissue remained. A few polymorphonuclear leucocytes appeared, and were followed in the next few days by macrophages. These were never present in great numbers.

The second stage began on about the fourth day after operation. In the adventitia, near the ends of the aorta, many capillaries and fibroblasts appeared. By the seventh day the fibroblasts were growing out from the aorta and beginning to replace the fibrin which was lining the prosthesis. Endothelium grew out with the fibrous tissue and even slightly ahead of it. It could be recognized on inspection as a shining pale surface. Figure II shows the naked-eye appearance during this stage. Figures III and IV illustrate the microscopic changes. It was also observed that on the seventh day capillaries and fibroblasts had arisen from the tissue on which the graft lay, and had also advanced from the adventitia of the aorta towards the outside of the nylon cloth. The angle between the outer surface of the nylon tube and the cut end of the aorta was invaded by granulation tissue first. At the same time fibroblasts advanced in to the necrotic areas of the aorta, which now appeared to be larger than before.

FIGURE II.

A prosthesis in the aorta after 51 days. The inner surface has been covered with rough fibrin. The shining tissue growing in from each end is fibrous tissue covered by endothelium.

The organization proceeded at different rates at different sites and in individual animals. In the inner surface of the prosthesis, tongues of fibrous tissue from each side had met in 29 days in one sheep. Another nylon tube examined after 44 days was almost completely lined by fibrous tissue and endothelium. In other animals the process was slower. In none was the replacement of this inner layer of fibrin complete, even though the last sheep was not killed until 137 days had elapsed. On the outer surface fibrosis was much faster. By 29 days much collagen had been laid down, and the nuclei of the fibrous tissue cells were resuming their thin resting shapes. The nylon material was now firmly bound to the surrounding tissue by connective tissue fibres which passed between strands of the yarn. By forty-four days organization was complete on the outside of the prosthesis. Some macrophages remained, especially at the ends of the arteries, and these were still present after 137 days. Some giant cells were seen near the sutures and against the threads of the material. Fibrosis of the necrotic areas of the aorta was complete in most animals by 51 days. Calcium was deposited in the necrotic areas in three sheep.

Discussion.

The Surgical Problem.

The perfect artificial arterial substitute is unlikely to be discovered, because the host provides only fibrous tissue to reinforce the prosthesis. Short of the ideal, the requirements of an arterial substitute fall into two categories, immediate and late.

Immediate requirements are as follows: (i) It must be readily available. (ii) It should be easily fashioned to various sizes. (iii) Sterilization must be easy. (iv) Insertion must not be too difficult. (v) It must be blood-tight, or rapidly become so. (vi) It must not cause any significant inflammatory reaction. (vii) It should be flexible. (viii) To be used peripherally, it must not be occluded by kinking at the flexures. The nylon filter fabric used in these experiments fulfilled all these requirements except the last.

The experiments do not show whether this nylon fabric will be satisfactory over a period of years. The late requirements are as follows: (i) The new arterial segment must not be specially prone to degeneration or calcification. (ii) The prosthetic material should last indefinitely, to prevent the theoretical danger that the new fibrous tissue may give way to form an aneurysm. (iii) It must not produce other hypothetical late ill-effects (*e.g.*, it must not be carcinogenic). It is mainly by the second group of criteria that arterial substitutes will be judged in the future.

The Anatomical Changes.

The weave of the nylon cloth was apparently too close to allow any significant number of fibroblasts to pass through from the outside. The fibrin layer on the inside of the prosthesis was replaced rather slowly by fibroblasts growing in from the ends of the aorta. No blood vessels were recognized with these fibroblasts. It may be noted here that Harris and his co-workers (1955), using prostheses of the same nylon filter fabric in dogs, found similar changes, except that rather more fibroblasts passed through the cloth from the outside. Blakemore and Voorhees (1954) used a prosthesis of "Vinyon-N" cloth. They found that fibroblasts invading the layer of fibrin had reached all parts of it by the fourth week. Endothelial proliferation was slower and not complete until between the seventh and fourteenth weeks.

The extensive necrosis of the muscle of the aortic media is presumed to be due to the loss of *vasa vasorum* and the local pressure of sutures. The elastic fibres did not appear to be affected at all. No stretching of the aortic wall was observed.

The polymorphs, macrophages and giant cells which were seen lay near necrotic areas, or about the nylon cloth or the suture material. There was no other inflammatory reaction and no calcification except in the aorta.

Summary.

A nylon filter fabric was tried as a substitute for a segment of the thoracic aorta in 17 sheep. In one experiment clotting occurred, causing death on the fourteenth day. The remaining prostheses remained patent. They were examined at various intervals. The initial deposit of fibrin on the inner surface was slowly replaced by fibrous tissue and endothelium growing out from the aorta. Fibrous tissue formed more rapidly outside the prosthesis.

These experiments justify the use of this nylon filter fabric for replacement of segments of the aorta.

Acknowledgements.

So many people played their part in this work that it would be impossible to make adequate acknowledgement individually. We are very grateful for their help. The photograph in Figure I was taken by Miss M. Simpson, of the Royal North Shore Hospital of Sydney. The other three photographs were taken by the Department of Illustration, University of Sydney.

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Addendum.

DURING the time that has elapsed since the completion of this work over two years ago, the use of synthetic materials has become widely accepted and numerous clinical reports have appeared in the literature.

Reports of Cases.

THE NATURE OF MALIGNANT TRANSFORMATION IN A CHRONIC ULCER.

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THE case to be described in this article is presented because of one striking feature, which appears to be worth discussing on account of certain scientific considerations and practical implications.

Clinical Record.

The patient, a woman, came under observation in March, 1952, at the age of 84 years. There was an ulcer on her left leg. Since the application of carbolic acid to the leg 50 years earlier this area had alternately ulcerated and healed. On the present occasion the ulcer seemed to the patient to be different. A biopsy was performed, and histological examination of sections showed the structure of squamous cell carcinoma. Amputation was advised, but the patient refused this form of treatment.

In April, 1952 (one month after original observation), the ulcer was excised. The specimen consisted of a piece of skin measuring 15 by 10 centimetres. The microscopic appearances were typical of squamous cell carcinoma. There was a good deal of dense fibrous tissue beneath the neoplastic tissue, and continuity of the surface epithelium with the neoplastic structure was readily seen. At the time of the excision the area was grafted with an autogenous split thickness graft.

In June, 1952 (three months after original observation), the graft appeared healthy except for two areas, from which biopsies were taken. As the microscopic appearances indicated that these areas were malignant, they were excised and the areas grafted as in April, 1952. The excised tissue consisted of two ellipses of skin measuring 3·5 by 2·0 centimetres and 4·0 by 2·5 centimetres respectively. The corresponding histological preparations (Figures I and II) show the structure of squamous cell carcinoma. In each instance the neoplasm can be traced in direct continuity from the surface layer of epithelium. This appearance is that seen in the case of a primary neoplasm which has not yet destroyed its own origins, but is rather surprising in the case of a "recurrence".

By August, 1952 (five months after original observation), two small ulcers had developed in the area, and in September (six months after original observation), an ellipse of tissue bearing the ulcers was excised. Two days later "postage stamp" grafting to the area was carried out. The ulcers measured 4·5 centimetres and 2·0 centimetres in diameter respectively. Both ulcers showed the histological features of squamous cell carcinoma, and in one of them (Figure III) direct continuity from the surface epithelium to the neoplastic tissue is clearly demonstrable.

In October, 1952 (seven months after original observation), carcinomatous activity was suspected in two more areas, and this was confirmed by histological examination of two excised ulcers, which measured 3·0 by 2·0 centimetres and 5·0 by 3·0 centimetres respectively. In one of these histological preparations, the same continuity from surface epithelium to neoplastic tissue is easily seen (Figure IV).

In January, 1953 (10 months after original observation), the patient was readmitted to hospital with two more ulcers within the area originally grafted. These were excised. They measured 6·0 and 3·5 centimetres in diameter respectively, and their corresponding histological features are shown in Figures V and VI. In both instances continuity from surface epithelium to neoplastic tissue was seen.

The topography of the areas was very carefully checked by Dr. E. G. Currow, Surgical Registrar, who had performed the grafting, and there was no doubt that all the ulcers were within the area of grafted skin.

By February, 1953 (11 months after original observation), there were further recurrences confirmed by histological study, and in April, 1953 (13 months after original observation), treatment by amputation was accepted by the patient. At the time of amputation a lymph gland was removed from the groin, and microscopic examination of sections revealed metastasis.

In August, 1953 (17 months after original observation), the patient suffered an episode of intestinal obstruction, seemingly due to incarceration in an old incisional hernia. This was successfully treated by intubation.

In July, 1954 (28 months after original observation) patient died in another hospital, at the age of 86 years, and no further details are available.

Discussion.

In six of the eight foci of recurrent neoplasia which were studied, the readily demonstrable continuity from the surface epithelium to the neoplastic tissue indicates that these carcinomatous foci were derived from the overlying epithelium, and not from portions of carcinoma left behind at the time of excision. Further, the overlying epithelium was grafted epithelium, and hence derived from a site remote from that damaged by the carbolic acid. The alternative interpretation, that residual carcinomatous tissue gave rise to the surface layer, seems too improbable for further comment.

Willis, in 1944 and 1945, reviewed evidence favouring the "field" origin of neoplasia. According to this notion, a noxious influence operates on a "field" of cells, and neoplastic transformation occurs over a considerable area rather than from mutation of a single cell. Morphological changes may sometimes suggest the existence of such a field of influence, as for example, in the case of a squamous cell carcinoma of the skin, the thickening of epidermis beyond the confines of the carcinoma or a greater degree of senile elastosis in the area. This does not mean, of course, that a field of influence cannot exist without morphological change.

The present case seems to demonstrate the existence of a carcinogenic influence emanating from the connective tissue damaged 50 years previously by carbolic acid.

Billingham *et alii* (1951) described experimental grafting, in which epithelium from skin areas in mice treated with the carcinogenic agent 20-methyl cholanthrene was transferred to untreated areas and epithelium from untreated areas was transferred to treated areas. In the former type of experiment carcinoma did not develop, whereas in the latter it did. Although interpretation of results was a little complicated by the presence of the deeper parts of the original hair follicles left in recipient areas, it seemed clear that the epithelium had not been acted upon directly by the 20-methyl cholanthrene but by the mediation of the connective tissue. This interpretation is in conformity with my interpretation of the case described. I am not aware of a previous record of this phenomenon in the human subject.

The local inadequacy of the surgical treatment in the human case would seem to reside in failure to remove exciting connective tissue rather than in failure to remove all of the already neoplastic epithelial tissue. There would seem to be no obvious means of recognizing the extent of this pathological change in the connective tissue.

It is interesting to note that although the connective tissue required 50 years to acquire its power of excitation, once this power was attained it could exert its specific effect on epithelium in the short space of a few months.

Summary.

A case is described which seems to be a clear demonstration of the fact that connective tissue may assume the power to excite squamous epithelium to malignant transformation.

It follows that adequate local surgical removal in such circumstances involves removal, not only of neoplasm, but also of exciting connective tissue, which may be more extensive than the epithelioma itself.

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PLEURO-PERITONEAL PASSAGE HERNIA.

By P. A. Tomlinson and L. Bernstein,
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Mrs. A. was admitted to the King George V Memorial Hospital, Sydney, in the eleventh week of her first pregnancy on December 24, 1955 (her eighteenth birthday). She said that six nights before, after turning over in bed, she had experienced severe upper abdominal pain and vomited. The following morning she went to see her obstetrician, complained mildly of some colic and constipation, and received opening medicine. It was arranged

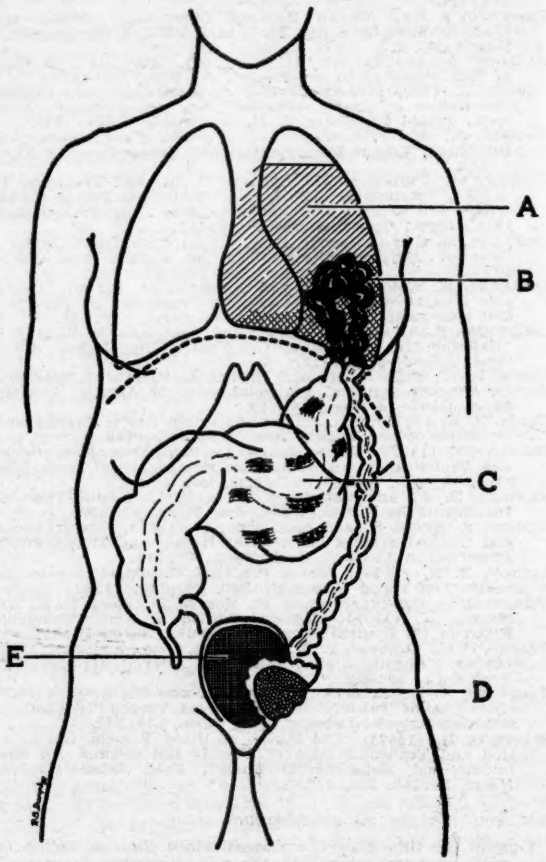

Figure I.

Findings at laparotomy. A, pyopneumothorax; B, ruptured gangrenous colon in the left pleural cavity; C, dilated proximal segment of colon with patches of gangrene; D, osteochondroma of pelvis; E, uterus with three months' pregnancy.

for a nurse to call and give her an enema. Over the next few days, the abdominal pain continued to recur, though she did not again report to her doctor. She had in addition severe constant pain behind the lower part of the sternum. Her abdomen became progressively distended, no bowel evacuation occurred despite a series of enemata, and there were bouts of vomiting. Breathing became more difficult and palpitations occurred. With her birthday and Christmas approaching, and not wishing to go to hospital, she held out at home till December 24, and then went to see her doctor again.

Mrs. A. had previously had a chondroma removed from the right humerus, and she was known to have multiple osteochondromata, one of which was large and rose from

the left side of the pelvis, encroaching on the cavity of the true pelvis.

Examination of the patient on her admission to hospital revealed her to be flushed, and to have a toxæmic appearance; her temperature was 98° F., and her heart rate was 116 per minute. Her abdomen was distended, and larger in size than a full-time pregnancy; it was tense, tender and tympanitic. Bowel sounds were present. It was evident that she had bowel obstruction. The left side of the chest was completely dull to percussion, and opaque on X-ray examination, with a horizonal upper border of opacity and some air above in the left pleural cavity. It was evident that the patient had (i) bowel obstruction and (ii) an effusion and pneumothorax on the left side, probably a pyopneumothorax.

The question of a diaphragmatic hernia was discussed. Intravenous therapy was commenced, the stomach was emptied by gastric tube, and anæsthesia was induced. It was decided to defer needling the left side of the chest till it was ascertained whether the patient was suffering from one pathological condition or two. If at laparotomy it was found that there was no connexion between the chest condition and the abdominal condition, then while the abdominal procedure was carried out, the chest could be aspirated by needle and evacuated by suction. Otherwise it was thought that the condition was primarily of abdominal origin, and might be managed from within the abdomen.

The findings at laparotomy (Figure I) were as follows: (i) There was a colonic obstruction at the left cupola of the diaphragm, through which a loop of bowel had passed into the left pleural cavity. (ii) The distal part of the colon emerging from the hole in the diaphragm was collapsed. (iii) The proximal portion of the colon was dilated to a diameter of six inches as far back as the caecum. (iv) Multiple patches of gangrene were seen in most of the transverse colon proximal to the site of the obstruction, but as yet no perforation within the abdomen. (v) Fourteen inches of gangrenous perforated colon were found in the left pleural cavity, with a fæcal pyopneumothorax. (vi) A large osteochondroma of the pelvis was present. At this stage the following became evident: (i) that resection of portion of the colon would have to take place; (ii) that resection and primary anastomosis could not be contemplated; (iii) that the pleural cavity contents were too thick to be emptied by needle alone; (iv) that the patient was too ill for major intercostal thoracotomy; (v) that it would be impossible to avoid contamination of the upper part of the abdomen with the thoracic empyema contents.

The intravenous administration of "Terramycin" was commenced. The diaphragm was incised laterally from the hernial orifice, and as much fluid as possible was aspirated from the chest with the sucker. The incision in the diaphragm was then increased to reveal greenish discoloration of the lower parietal part of the chest wall. The gangrenous bowel was reduced into the abdomen. An intercostal tube was inserted into the left lower quadrant of the chest and connected to an underwater drain. The hole in the diaphragm was repaired with chromicized gut. The gangrenous colon, including the transverse colon, was resected. The ends of the colon remaining were brought out through separate stab wounds, in the left and right hypochondria. The left side of the colon was not mobilized and brought out with the right (as a Mikulicz procedure), partly because the patient was too ill, and partly to avoid opening up any further raw surfaces for contamination.

The post-operative treatment was as follows. The abdomen required no attention other than removal of the bowel clamps. Under-water drainage of the chest was continued till January 12, 1956, and open drainage till January 23. Gastric drainage was maintained until bowel sounds returned on the second day. "Terramycin" was given, and sulphonamides were administered by the intravenous route.

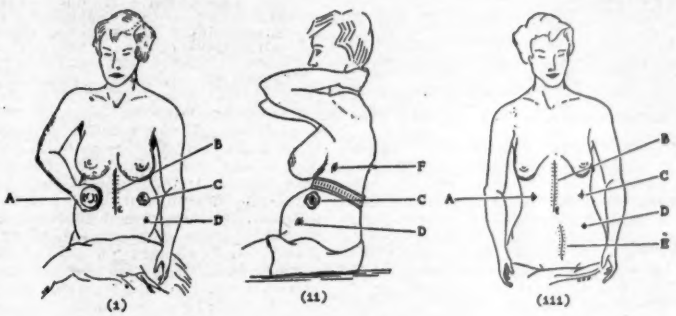

FIGURE II.

(i) at the seventh month; (ii) at the seventh month; (iii) after restoration of the continuity of the bowel. A, functioning colostomy site; B, laparotomy scar; C, vent to left side of colon; D, site of drainage of abdominal cavity; E, Cæsarean section scar; F, site of drainage of pleural cavity.

Fluids were also given intravenously, according to the serum electrolytes. "Cortef" was given on the third and fourth days, and strychnine by injection. Gas-gangrene antiserum, antitetanus serum, vitamin B_1 and vitamin C were also given. The colostomy functioned on December 26, 1955, the second day after operation.

The patient's survival was at first in doubt. Her pulse rate was between 240 and 200 per minute all Christmas Day and Boxing Day, and then settled gradually; but it remained persistently high at about 130 per minute for some 14 days. X-ray and electrocardiographic examination revealed no cardiac enlargement and no signs of toxic myocarditis, but sinus tachycardia. Her improvement was thereafter progressive. The empyema cavity diminished to a mere track, and the intercostal drainage tube was then removed, the lung being fully expanded.

On February 10, her husband's birthday, the patient left hospital wearing a colostomy bag over the functioning right-sided colostomy, and a pad over the opening into the left side of the colon. Further surgery was withheld until the child was delivered and established. Her subsequent course was without complication apart from slight occasional prolapse of the colostomy, and early in July, 1956, the patient was readmitted to hospital and had her baby by Cæsarean section (on the writer's birthday). It was suggested that the chondroma should be removed at this time, but the patient declined. Examination of the colon, immediately after the uterus had been emptied, indicated that there was sufficient length of colon available to restore continuity without difficulty. It was decided to let the patient carry on with her colostomy until the baby was weaned.

Mrs. A. was readmitted to the Royal Prince Alfred Hospital on October 22 for restoration of the continuity of the colon. At operation the left side of the colon was mobilized and brought out beside to the functioning colostomy, a spur being sewn as a Mikulicz type of procedure. This spur was later crushed and the colostomy was closed. The patient

left hospital on November 23, again declining to have the osteochondroma removed. The baby is quite well and of normal development.

Summary.

A case is reported of a strangulated hernia through the left pleuro-peritoneal passage of 14 inches of gangrenous colon (with perforation and pyopneumothorax); a three-month gestation survived in this patient, who also had multiple osteochondromata encroaching on the cavity of the true pelvis.

Acknowledgements.

Thanks are due to members of the resident staff of King George V Memorial Hospital and the Royal Prince Alfred Hospital, especially Dr. Jean Collison, who administered the first anæsthetic, and Dr. L. Bernstein, who was responsible for most of the post-operative treatment.

Reviews.

Traquair's Clinical Perimetry. By G. I. Scott, M.A., M.B., F.R.C.S. (Ed.), with a foreword by Norman M. Dott, C.B.E., M.B., F.R.C.S. (Ed.); Seventh Edition; 1957. London: Henry Kimpton. 9⅝" × 7¼", pp. 354, with 274 illustrations. Price: 60s.

For slightly more than 30 years "Traquair's Clinical Perimetry" has been the standard reference book on fields of vision. Eight years have passed since the sixth edition, and of late there have been other books by British and American authors, one of which set a new standard in illustration; but there has been nothing to replace "Traquair" in authority. With this new edition by G. I. Scott, we again have the outstanding work on the subject under the same title. Scott has preserved most of the original text, has added to it considerably, and has modernized and amplified the illustrations, a large number of which are entirely new. Essentially the book remains in spirit and content as it was—one more tribute to the permanence of the honest, painstaking and valid observation on which Traquair spent his life. It is a tribute, too, to the present author, who has managed to keep all the best of "Traquair" while adding, in the same careful and authentic spirit, valuable examples of his own, as well as up-to-date material on equipment, theory and the interpretation of fields.

There are some improvements, too; the size of the page is more convenient than the old, the print seems better and the illustrations and legends are bolder.

There is perhaps no book more packed with "meat" than this; hardly a word is superfluous, opinions and authority are weighty, and facts come one upon another so fast that reading must be meticulous and intermittent to gain all it contains.

"Traquair's Clinical Perimetry" is still the classic of ophthalmology, and G. I. Scott is a worthy successor to one of the greatest men of ophthalmology. Moreover, the price of the book, in these days and in comparison with that of other recent works on the same subject, is reasonable indeed.

Stedman's Medical Dictionary. Edited by Norman Burke Taylor, V.D., M.D., F.R.S.C., F.R.C.S. (Edin.), F.R.C.P. (Can.), M.R.C.S. (Lon.), in collaboration with Lieutenant-Colonel Allen Ellsworth Taylor, D.S.O., M.A.; Nineteenth Revised Edition; 1957. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9¾" × 6¾", pp. 2704, with many illustrations. Price: £6 17s. 6d.

To most people the compiling of dictionaries would seem a tedious occupation, and it therefore seems rather surprising that there should be at least three major medical dictionaries published in America, all more or less equally bulky and comprehensive. This new edition of "Stedman's Medical Dictionary" makes it for the moment the most recent of the trio. The four years that have passed since it was last revised have been prolific in the creation of new medical terms, and the editors state that several thousand of these have been included in the present edition. Some new illustrations have been added, and many of the old ones redrawn. These are mainly small line drawings, which are liberally scattered through the text; whether they are really of much assistance depends perhaps on the purpose for which the dictionary is being used; other dictionaries have largely or entirely dispensed with them. Structural formulæ accompany the description of chemical compounds wherever necessary. There is an introductory section of 45 pages on medical etymology, and the pretensions to a classical bias are further evidenced by the inclusion of the names of many early medical authorities. There are some useful appendices at the back, but the bulk of this section is taken up by sixty pages on anatomical nomenclature. The table giving Basle anatomical nomenclature equivalents of the terms in current English usage may be useful to those who have studied anatomy in European countries. The *nomina anatomica* adopted by the International Anatomical Nomenclature Committee in 1950 are also given in full.

This dictionary has a number of useful features, and features which may appeal to some but not to others. It contains a few excessively obscure and obsolete terms, but fails to include certain others in current usage, and the final impression is that both the selection of material and its presentation fall short of the highest standard. The type is very small, but the items are well spaced.

Children No Longer: A Practical Guide on Understanding the Adolescent. Guide Series No. 7; Second Edition; 1957. Sydney, Melbourne and Brisbane: The Father and Son Welfare Movement of Australia. 7¼" × 5¾", pp. 44, with illustrations. Price not stated.

Parents of adolescent boys and girls will find this little book of real help, not only in the direct guidance of their children, but also in determining their own attitude and behaviour. Written by a practising psychiatrist, it contains a great deal of common sense and aims at the cultivation of an understanding attitude. It deals successively with physical development, psychological development and spiritual development, and covers a surprising amount of ground. The treatment of some subjects is very brief, but probably quite enough is said on each, and the result is a book small enough and straightforward enough to be read by any busy parent of average intelligence. The section on spiritual development is essentially Christian in its character, but it should be acceptable to most people in this country. Doctors looking for a guide to put into the hands of puzzled parents should find this little book a great help.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Peter T. Bohan Memorial Lectures on Medicine"; First Series; 1957. Lawrence: University of Kansas Press. 9¼" × 6", pp. 152, with four illustrations. Price: $3.00.

Seven lectures on various clinical subjects.

"Biochemical Investigations in Diagnosis and Treatment", by John D. N. Nabarro, M.D., F.R.C.P.; Second Edition; 1958. London: H. K. Lewis and Co. Ltd. 7⅞" × 5¼", pp. 311, with four illustrations. Price: £1 5s. (English).

The first edition was published in 1954.

"Blacklock and Southwell: A Guide to Human Parasitology for Medical Practitioners", revised by T. H. Davey, O.B.E., M.D., D.T.M.; Sixth Edition; 1958. London: H. K. Lewis and Co. Ltd. 9¼" × 5⅝", pp. 230, with three coloured plates and 119 illustrations. Price: £1 10s. (English).

For the practitioner who sometimes has to make a diagnosis in diseases caused by animal parasites, and for those taking diploma courses.

"Introduction to Clinical Endocrinology", by A. Stuart Mason, M.A., M.D., B.Ch., M.R.C.S., M.R.C.P.; 1957. Oxford: Blackwell Scientific Publications. 8½" × 5¼", pp. 200. Price: 22s. 6d. (English).

An attempt to present clinical endocrinology in terms of applied physiology.

"The Prevention and Control of Infection in the Newly Born and the Care of the Premature Baby"; 1958. Sydney: Department of Public Health, Division of Maternal and Baby Welfare. 8¼" × 5¼", pp. 52. Price not stated.

Contains sections on general planning for obstetric units and nurseries, on staphylococcal infection (prevention and control) and on care of premature babies.

The Medical Journal of Australia

SATURDAY, APRIL 5, 1958.

ACUTE PANCREATITIS.

ACUTE PANCREATITIS is still a baffling disease from whatever aspect it is viewed. Its causation is uncertain, its diagnosis difficult, its treatment far from specific, and its prognosis in those surviving doubtful, to say the least. Nevertheless, new experimental and clinical work is gradually helping to resolve many of its perplexities, and it is of interest to review this.

There are undoubtedly many and varied ætiological factors in the production of this disease. R. Joshi, J. Probstein and H. Blumenthal[1] point out that in two-thirds of the cases the ætiology is unknown. These same authors in another paper[2] mention that the ætiological factors of acute pancreatitis may be grouped into obstruction of the common bile and/or pancreatic ducts, dietary deficiencies, the ingestion of toxic substances, especially alcohol, viral or bacterial infections, allergic factors, trauma and vascular factors. They discuss 12 autopsy cases of atheromatous embolization from the aorta to pancreatic arteries, in all but two of which there was an associated acute pancreatitis of varying intensity. The significant feature was that no other causal factor, such as an obstructed pancreatic duct, was present in any of these cases.

Puestow, in a paper presented at the fourteenth annual meeting of the Central Surgical Association in Chicago in February, 1957 (quoted by L. Dragstedt[3]), described a series of patients with relapsing pancreatitis treated successfully by retrograde drainage of the pancreas into the intestine. Catheter studies using radiopaque material in the pancreatic duct showed that in most of these cases the distal end of the main duct was stenosed, often over three to five centimetres. The ducts proximal to this were much dilated, and the body and tail of the pancreas were fibrotic. It may be wondered perhaps why the communication between the accessory duct of Santorini and the main pancreatic duct might not enlarge to act as a "safety valve" for the dilated main duct; but, as pointed out by I. Lichtenstein,[4] an appreciable communication is present in only 15% of human subjects. In discussing Puestow's contribution, Dragstedt writes that one could hardly doubt that the pancreatic duct obstruction was the cause of the necrosis of the pancreas with its fibrous replacement and also of the recurrent pain characteristic of this disease.

[1] *Am. Surgeon*, 1957, 23 : 34.
[2] *Arch. Surg.*, 1957, 75 : 566.
[3] *Arch. Surg.*, 1957, 75 : 581.
[4] *Gastro-enterology*, 1957, 33 : 64.

Dragstedt points out that the essential feature in pancreatitis is necrosis, and that more extensive necrosis occurs when the pancreas is actively secreting. This is confirmed by the observations of W. Wurma,[1] who found that the onset of symptoms frequently followed a fat-rich meal in a series of 248 patients. Dragstedt considers that, whatever may be the cause of the initial lesion of acute pancreatitis, whether it is pancreatic arterial embolization, obstruction to the pancreatic duct, or the passage of bile into the secreting pancreas, or a combination of these and other factors, the end result is determined by the rapidity and extent of this necrosis. He considers that bile passing into an actively secreting pancreas and extensive vascular damage to the pancreas are the main causes of acute pancreatitis sufficiently severe to cause death. On the other hand, he thinks that relapsing pancreatitis is probably due to intermittent obstruction of the pancreatic duct causing also pancreatic duct dilatation. He points out that ligation of the duct, as in the experimental dog or in patients who have had the head of the pancreas removed, causes gradual necrosis and replacement fibrosis but no acute pancreatitis or dilatation of the remaining ducts.

The passage of bile into the pancreatic duct in the actively secreting pancreas provides an explanation for the high incidence of acute pancreatitis in patients with antecedent gall-stones in the common duct or gall-bladder. However, in most cases, because of the absence of a gall-stone impacted in the ampulla of Vater, it is assumed that adoption of the common channel must be due to spasm of the sphincter of Oddi. Such spasm due to traumatic œdema after sphincterotomy of the ampulla may be the cause of the complicating acute pancreatitis apt to follow this operation (M. Roux and R. Rittori[2]), as also the acute pancreatitis apt to follow choledocho-lithotomy, especially when a long-arm T-tube of the Catteil type is used and inserted across the sphincter (J. Thompson, J. Howard and K. Vowles[3]).

These postulations, of course, take no account of the work of Hilary Long,[4] who showed that the sphincter of Oddi (*sphincter choledochus inferior*) does not surround the pancreatic duct, nor do they take note of the observations of Ian Aird[5] that acute pancreatitis can occur solely in the wedge of pancreas drained by the accessory duct of Santorini, or in glands whose main ducts drain separately into the duodenum from the common bile duct or whose main duct is replaced by the duct of Santorini.

Local trauma to the pancreas, as may occur during a gastrectomy, can explain acute pancreatitis following this operation, but not pancreatitis after operation on organs elsewhere, such as a prostatectomy. Arterial embolization in these usually elderly patients may be the explanation. It is hard to see why the ingestion of alcohol should cause pancreatitis, especially recurrent attacks, with or without associated biliary tract lesions. It is sufficient to say that, because of the frequency of this ætiological factor, all patients subject to recurrent pancreatitis should become strict teetotallers. It is hard also to see why the giving of

[1] *Deutsche med. Wchnschr.*, 1957, 82 : 75.
[2] *J. chir.*, 1956, 72 : 76.
[3] *Surg., Gynec. & Obst.*, 1957, 105 : 706.
[4] *Brit. J. Surg.*, 1942, 29 : 422.
[5] "Companion in Surgical Studies", 2nd edition, 1957, Livingstone : 1013.

cholopaque substances to patients with jaundice should lead to acute pancreatitis, as has been suggested, especially since in these persons the dye seems to be mostly excreted by the kidneys, as witness the resultant excellent pyelograms obtained in such patients. The virus of mumps can produce a very severe form of acute pancreatitis, as anyone can testify who witnessed mumps epidemics in the army in the second World War, when this complication was common.

The advent of cortisone therapy has produced further complicating factors in the ætiology of this disease. F. Carone and A. Liebow[1] studied sections of the pancreas obtained at autopsy from 54 patients who had been treated with cortisone, prednisone, prednisolone or corticotrophin. Evidence of patchy acute pancreatitis was present in 16 cases, but in only three was the evidence severe. The pancreatitis was mainly intestinal, and necrosis of the acinar cells and hæmorrhage were not prominent. In addition, ectasia of the pancreatic acini was present in 32 of their 54 cases (though in 13 of 54 untreated control cases it was also present). It is to be noted that none of the patients had symptoms of this disease during life. In contrast, H. Baar and O. Wolff[2] reported two cases of death from symptomatic acute hæmorrhagic pancreatitis in children treated by cortisone for other disorders. Such findings must now make one very wary of treating acute pancreatitis with cortisone, as has been done in the past, even though favourable results were reported (J. Solem, O. Knutrud and K. Andresen[3]).

There is little new so far as diagnosis is concerned, except that it can still be very difficult. In a series of 248 cases reviewed from 1920 to 1954, W. Wurma[4] found that only 4% were diagnosed correctly on the patient's admission to hospital. All patients with equivocal upper abdominal pain should have a serum amylase estimation performed, it being remembered that this may be raised above normal in many lesions other than acute pancreatitis; the most recently implicated is acute intestinal obstruction in the experimental dog (J. Byrne and J. Boyd[5]).

All clinicians agree on conservative non-operative treatment for an attack of acute pancreatitis, provided the diagnosis is certain. D. Elliott[6] emphasizes that significant deficits in whole blood volume appear early in the course of severe acute pancreatitis whenever there is significant ileus, and he advises early replacement by the liberal use of whole blood and albumin; this, he considers, has reduced the over-all mortality in all forms of acute pancreatitis from 19·5% in 1952 to 6·5% in patients treated subsequently at the Ohio State University College of Medicine.

The prognosis is uncertain, as acute pancreatitis is a recurrent disease. T. Rose[7] showed that, of a series of 69 patients with acute pancreatitis, 19 died in the first attack or shortly afterwards, and six others during a period of up to 23 years. Of 30 patients followed longer than twelve months, 24 had at least one further acute attack, some had as many as eight attacks over a period of 16 years, and only six had no further attack in a period up to as long as 23 years. Of the 24 patients with acute relapsing pancreatitis, eight showed symptoms characteristic of chronic pancreatitis. Van G. Kaden and J. Howard[1] followed 100 patients with acute pancreatitis, who were mainly diagnosed clinically as they were managed conservatively without definite therapy. Recurrences occurred in 39 cases. They distinguished two patterns of recurrence, depending on the presence or absence of demonstrable biliary tract abnormality. Of patients with normal gall-bladders, 16% developed pancreatic calcification and 8% pseudocysts. In no patients with gall-stones did a pseudocyst develop. Of the patients with gall-stones, only 3% of those who had an operation for gall-bladder disease had recurrences. J. Howard and G. Jordan, junior,[2] state that definite gall-bladder surgery appears to interrupt the course of associated pancreatitis if biliary tract disease is present. Removal of the normal gall-bladder does not appear to influence the incidence of recurrence.

[1] *New England J. Med.*, 1957, 257: 690.
[2] *Lancet*, 1957, 1: 812.
[3] *Acta chir. scandinav.*, 1955, 109: 415.
[4] *Deutsche med. Wchnschr.*, 1957, 82: 475.
[5] *New England J. Med.*, 1957, 256: 1176.
[6] *Arch. Surg.*, 1957, 75: 573.
[7] M. J. Australia, 1951, 2: 453.

[1] *Arch. Surg.*, 1956, 73: 269.
[2] *Arch. Surg.*, 1956, 73: 960.
[3] *Brit. M. J.*, 1953, 1: 799 (April 11).
[4] *Brit. J. Prevent. & Soc. M.*, 1957, 11: 107 (July).

Current Comment.

BLOOD GROUPS AND DISEASE.

For centuries we have spoken of diathesis, particularly referring to inborn susceptibility to disease, but it has not been until this century that through the study of genetics the term has acquired real meaning. The study of twins has been an accredited method of genetic research for many years, and this journal has recently published valuable information along these lines from Melbourne. It is disappointing to learn that this work, some of which is still to be published, was brought to a close at the end of 1956 because, in the words of the 1957 Nuffield Foundation Report, "it proved impossible for a small group of workers to obtain sufficient material under Australian conditions".

Blood groups are possibly the most direct genetic differences between men which can be defined, and with a large body of data relating to some blood groups available from hospital records where diseases are also detailed, it has been possible in the last few years to put the hypothesis of diathesis to further scrutiny. Although abortive attempts in this direction were commenced in the twenties, no uniformly acceptable results were obtained until the matter was brought to prominence largely through the work of I. Aird, H. H. Bentall and J. A. Fraser Roberts of England in 1953,[3] who showed an association between blood group A and carcinoma of the stomach. Subsequently there have been further claims and denials in regard to other diseases, and Fraser Roberts has recently reviewed the available data[4] showing that in fact some of the previous workers were closer to the mark than had been realized. Information relating to A B O and Rh blood groups are readily available, and other blood group and genetic differences between individuals are being studied prospectively now that the hypothesis can be considered proven.

In some diseases, notably those of the upper alimentary tract, the evidence accumulated from several countries is clear cut. The risk of developing a duodenal ulcer appears to be 40% higher in individuals belonging to blood group O than in individuals of other A B O blood groups. Gastric ulcer also occurs more frequently in individuals of blood

group O than can be explained by chance, although the degree of association is not as great as in duodenal ulcer. Evidence at present available suggests that individuals of blood group O run a higher risk of developing stomal ulceration after surgical treatment for duodenal ulcer. Both pernicious anæmia and cancer of the stomach have a higher relative incidence in individuals of blood group A. Clear-cut negative results with regard to the A B O system have been obtained in cancer of the colon, rectum, breast and bronchus, pulmonary tuberculosis, rheumatic fever, hypertension and toxæmia of pregnancy. Results indicating a field for further investigation have suggested associations between blood group A and *diabetes mellitus*, cirrhosis of the liver and infants dying of bronchopneumonia, and between blood group O and pituitary adenomata. No definite associations with the Rhesus system have yet been obtained.

It seems likely on the evidence available that the associations between diseases and blood groups are not the result of incomplete miscegenation in the population, but are a manifestation of R. A. Fisher's dictum[1] that no gene is neutral in natural selection; and in this connexion A. C. Allison's demonstration[2] that the sickle cell trait confers some degree of resistance to subtertian malaria is significant. The explanation of the association between A B O blood groups and gastro-duodenal diseases has received closer attention. It is known that individuals can be classified upon a genetic basis into those who secrete the specific A B O blood group mucoids into their saliva, gastric juice and elsewhere (secretors) and those who do not (non-secretors). C. A. Clarke and others of Liverpool[3] have shown that the characteristic of non-secretion also increases the risk of developing a duodenal ulcer. With the recent knowledge, reported by L. E. Glynn, E. J. Holborow and G. D. Johnson,[4] that the different histological types of gastric and duodenal mucosa also differ in their capacity to secrete blood group substances, a complex picture results, and it seems not improbable that the associations between gastro-duodenal diseases and the A B O blood groups may have an explanation at the physiological level.

Although these discoveries do not affect clinical practice, they represent one application of genetics and biometrics to common human diseases which so far have not been easily studied by these methods. With the growth of departments of clinical genetics at several medical centres, and with many other blood groups to act as genetic markers, further developments are awaited with interest.

THE BREASTS AND BREAST FEEDING.

IT is a matter of regret to many who knew Harold Waller personally that he wrote so little, and that his work was not more widely known. However, in a little volume published after his death,[5] he has given the fruits of a lifetime's work in the service of mothers and babies at Woolwich Hospital. It is clear and concise and well illustrated and should find its way onto the shelves of all doctors and nurses interested in the feeding of babies.

The failure of breast feeding is so common in civilized countries that the majority of doctors now rationalize the situation and regard artificial feeding as a scientific advance on breast feeding. However, the fact remains that mothers do want to feed their babies, and psychiatrists are rallying to the cause and stressing the need for improved relationships between mothers and their babies. This relationship, good or bad, has its origin in the attitude and experience of the mother in the pre-natal and immediate post-natal period. MacKeith and his co-workers have confirmed Waller's finding that skilled supervision of the mother in these periods will double her chance of feeding her baby normally. Whereas a statistical study conducted in 1948 by the Royal College of Obstetricians and Gynæcologists and the Population Investigation Committee revealed that 43% of women in Britain had ceased to breast feed their infants by the eight post-partum week, Waller obtained 90% successful breast feeding.

Waller was not interested in psychiatry, but his methods are good psychologically. This book deals only with techniques; it describes ante-natal preparation and doctor and midwife supervision in the maternity hospital period, breast anatomy and physiology and breast infections. The only criticism is one that must apply to every text-book—namely, that therapy soon becomes dated. Most doctors will hold that penicillin has now lost its efficacy in breast infections and should not be used, that mercury ointments are inadvisable for cracked nipples, and that the doses of stilbœstrol are too large. Waller always used large doses of stilbœstrol; but he kept his patients in hospital up to three weeks and was able to see their milk supply return after the initial depression. Under our conditions, smaller doses must be used for a shorter period for engorgement, and the use of stilbœstrol is generally avoidable in breast infections.

However, these are but minor criticisms, when one realizes how much Waller has added to our knowledge of the management of lactation by his careful clinical observations of the living human patients to whom he devoted his life.

RADIOACTIVE FAT IN ABSORPTION STUDIES.

UNTIL recently tests used to detect abnormalities in fat absorption were difficult to perform. Since the introduction of the use of I^{131} labelled triolein, by S. J. Thannhauser and M. M. Stanley[1] in 1949, many papers have appeared on the subject. D. Berkowitz and D. Sklaroff[2] have simplified the test for use in a large hospital with few laboratory facilities. All the apparatus needed is a scintillation well counter. The patient is given a dose of peanut oil emulsified with water with the use of Tween 80 and containing an amount of I^{131} labelled glycerin trioleate depending on the weight of the subject. Venous blood samples are then taken until a peak radioactivity level has been reached. With normal persons the peak blood level occurred most frequently between the fifth and sixth hours and was approximately 16% of the dose given. In five cases of sprue the isotope curve was flat in each case. In ten cases of chronic pancreatitis, confirmed by operation, the curve was significantly lower than normal. Other gastro-intestinal diseases showed little departure from normal. In cirrhosis of the liver the curve was normal. Patients with extrahepatic jaundice were, however, sharply distinguished from those with non-obstructive jaundice. In the extrahepatic jaundice the isotopic curve was much lowered, but this was not so in non-obstructive jaundice.

In the interpretation of data Berkowitz and Sklaroff, who have examined 200 patients, consider that four factors are of importance: (i) the sum of the fourth, fifth and sixth hour blood radioactivity values; (ii) the time at which peak absorption occurs; (iii) the magnitude of the peak; (iv) the rate of disappearance of activity from the blood. The fourth-to-sixth-hour absorption sum is a convenient way to compare results on a semiquantitative basis. A low or flat absorption curve when associated with a subnormal peak value has invariably been indicative of an abnormal state. It is possible by the use of this test to determine the improvement in fat absorption after treatment, as, for example, in sprue after corticoid therapy. This is but another example of the increasing use of isotopes in medicine and of the necessity for hospital laboratories to have the necessary equipment and trained personnel.

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[1] "The Genetical Theory of Natural Selection", 1930, Clarendon Press, Oxford.

[2] *Brit. M. J.*, 1954, 1: 290 (February 6).

[3] *Brit. M. J.*, 1956, 2: 725 (September 29).

[4] *Lancet*, 1957, 2: 1083 (November 30).

[5] "The Breasts and Breast Feeding", by Harold Waller, M.B., B.Ch. (Cantab.), F.R.C.O.G.; 1957. London: William Heinemann (Medical Books), Limited. 7¼" × 4¼", pp. 64, with 16 illustrations. Price: 7s. 6d.

[1] *Tr. A. Am. Physicians*, 1949, 62: 245.

[2] *Arch. Int. Med.*, 1957, 100: 351 (December).

Abstracts from Medical Literature.

OBSTETRICS AND GYNÆCOLOGY.

Labour Complications and Cerebral Palsy.

R. W. Fuldner (*Am. J. Obst. & Gynec.*, July, 1957) presents the results of a study of obstetrical factors in the ætiology of cerebral palsy. The clinical material consisted of 204 children with cerebral palsy examined at the Newington Home and Hospital for Crippled Children, Newington, Connecticut, over the preceding six years. The method used was a statistical study of the hospital records of those children. The author points out that opinion is divided on the role of abnormal labour; a striking incidence of labour complications in his series of cases led to the study. He concludes that a tentative correlation, chiefly as a matter of clinical interest, can be set up between certain of the chief complications of labour and one or more types of cerebral palsy. No inevitable relationship between cerebral palsy and a given complication of labour is implied. Clinically, it is necessary to think in terms of a progression of causes which finally exceeds the limit of fœtal tolerance. This point requires mention because the potential significance of complicated labour sometimes seems diminished when a group of infants can be shown to have survived it largely unscathed. The results of the study, from the obstetrical standpoint, are summarized in a tentative series of correlates. The author states that the correlates are not meant to be exclusive from group to group; to some extent all forms of cerebral palsy share the same background. The correlates are as follows: (i) Athetoid quadriplegia: persistent malposition, breech presentation, cord encirclement, arrest of labour, middle or high application of forceps. (ii) Spastic quadriplegia: prematurity, breech presentation, detachment of the placenta. (iii) Spastic hemiplegia: cord encirclement, maternal illness. (iv) Spastic paraplegia: prematurity, breech presentation, detachment of the placenta. The author shortly discusses the pathogenic mechanism in cerebral palsy, and concludes that in the majority of cases the primary cause is cerebral anoxia.

Fœtal Death in Utero.

V. Tricomi and S. G. Kohl (*Am. J. Obst. & Gynec.*, November, 1957) have investigated intrauterine fœtal death. Their study is based on a series of 16,057 deliveries in the King's County Hospital from January 1, 1951, to June 30, 1955. All the infants weighed over 1000 grammes, and there were 165 cases of fœtal death prior to the onset of labour, an incidence of 1·03%. The findings were as follows: There was no difference between the patient's history and the physical examination with regard to fixing the time of fœtal death. The patients in the study group had an older age distribution that the ordinary clinic population, though there was no difference in parity. Of the fœtuses, 7% were retained for 22 days or more; the longest period of retention was 120 days. There was no difference in the incidence of post-partum hæmorrhage between the study group and the clinic at large. Premature separation of the placenta was associated with 32% of the fœtal deaths, toxæmia was implicated in 21%, and diabetes was involved in 11·5%; a true knot of the umbilical cord was encountered in three instances. In none of these cases was syphilis the cause of death. Of the fœtal deaths, 36% were thought to be preventable; the responsibility was equally divided between patient and physician. There was one maternal death, which followed spontaneous rupture of the uterus prior to labour. The authors conclude that the results of their study support the concept of conservative management and abstinence from interference in cases of prolonged retention (more than 21 days) of a dead fœtus.

The Stein-Leventhal Syndrome.

R. P. Ellett and D. D. Barnes (*Am. J. Obst. & Gynec.*, December, 1957) review the current literature and present case reports of the Stein-Leventhal syndrome, which was first described in 1935 and correlated bilateral polycystic ovaries with a clinical syndrome of menstrual irregularity, especially amenorrhœa, and a history of sterility. Sterility is usually primary, but may develop subsequent to a pregnancy, and hirsutism occurs in approximately one-half of these women; obesity is fairly frequent. The diagnosis is made mainly on the history and physical findings, but Stein and his co-workers used pneumo-radiography in over 90 patients. Culdoscopy has been used by other workers. The most satisfactory treatment for the syndrome appears to be wedge resection of the ovaries. The remaining follicle cysts are perforated and the defect is closed with fine catgut. In Stein's series 57 out of 64 infertile patients became pregnant one or more times. Following wedge resection of the ovaries, hypoplasia of the uterus and underdevelopment of the breasts are usually corrected in a short period of time. The menstrual periods are usually resumed within five weeks following the operation and as a rule remain regular. Pregnancy usually occurs within a period of five months. If pregnancy does not occur within a short period of time, the likelihood of conception is remote. Hirsutism, if present, generally persists.

Perinatal Mortality of Cæsarean Section.

C. A. Gordon (*Am. J. Obst. & Gynec.*, November, 1957) presents a study of 2789 cases of Cæsarean section which were associated with 97 neonatal deaths and 58 fœtal deaths. The perinatal mortality rate was 55 per thousand. The obstetrical indications for Cæsarean section in those cases associated with perinatal death were dystocia, toxæmia, previous Cæsarean section and ante-partum hæmorrhage. Of the perinatal deaths, 14% were associated with dystocia and ante-partum hæmorrhage preceded perinatal death in 46%. There was a history of previous Cæsarean section in 41 cases of perinatal death, or 26% of the total. Prematurity was an important factor, as more than half of the operations were performed before the thirty-sixth week. The author states that perinatal death due to dystocia is largely controllable. Birth trauma associated with protracted labour, trial forceps and scapular presentation can be avoided. In cases of previous Cæsarean section prematurity is often added to the hazards of the fœtus, and the best time for operation is as close to term as possible, certainly later than 38 weeks. The premature baby with hæmolytic disease does badly and is prone to kernicterus. Early termination of pregnancy may be justified, but not by Cæsarean section. In cases of accidental hæmorrhage even slight placental separation may be attended by high fœtal mortality. If labour does not progress smoothly and rapidly after rupture of the membranes and uterine stimulation, earlier Cæsarean section should reduce fœtal mortality. Expectant treatment in *placenta prævia* produces the best results and the patient should remain in hospital up to the thirty-sixth week or later, when operation may be performed. As incision through the placenta will cause severe fœtal blood loss and increase the mortality rate, lower segment Cæsarean section should not be performed for *placenta prævia*.

Tuberculosis and Pregnancy.

G. Schaefer, S. J. Birnbaum and R. G. Douglas (*J.A.M.A.*, December 28, 1957) report on the present-day treatment of tuberculosis and pregnancy at the New York Lying-In Hospital. The incidence of tuberculosis complicating pregnancy at this hospital has remained between 1·5% and 2% of all ante-partum admissions from 1933 to 1951. Complete bed rest is no longer used as the sole treatment for the pregnant patient with tuberculosis, but modified bed rest is still advised during pregnancy. Pneumothorax is now rarely used as a means of collapse therapy in these patients. It has been displaced by chemotherapy and major thoracic surgery. From 1933 to 1951 only 1% of deliveries received antimicrobial drugs, compared with 37% of the deliveries from 1952 to 1956. The drugs used were streptomycin, amino-salicylic acid and isoniazid. The dosage and combination of these drugs were identical to those used in non-pregnant patients, and no untoward effects were noted in the mother or newborn infant. In the 1952 to 1956 series major thoracic surgery was performed on 11% of the patients before or during pregnancy, compared with 2% in the previous series. The present trend is towards excisional surgery, that is lobectomy, pneumonectomy or segmental resection rather than collapse procedures. Surgery may be performed during pregnancy. In 3·7% of cases there was a progression of disease after delivery, in 86% there was no change, and in 10% there was improvement. Excessive analgesia and anæsthesia should be avoided in the tuberculous patient. Local infiltration and pudendal block are preferred for anæsthesia. Vaginal delivery, with use of forceps to shorten the second stage when indicated, is the method of delivery. The authors note that since the dose of röntgens delivered in taking a conventional 14 by 17-inch

chest X-ray film is one-fiftieth of that delivered by the screening technique, the former method has superseded the latter.

Puerperal Breast Abscess.

C. O. SMITH AND A. VARGA (*Am. J. Obst. & Gynec.*, December, 1957) report on the cases of puerperal breast abscess occurring at the Cook County Hospital in the past decade. Although the number of deliveries has increased approximately three and one-half times, the number of abscesses has increased approximately eight and one-half times. In this series the commonest organism isolated was pathogenic *Staphylococcus aureus*, and although penicillin in large doses apparently produced good clinical response in 75% of patients, sensitivity tests showed resistance to penicillin in 89% of cases, suggesting that this therapy is of little or no value. Routine cultures on admission and discharge of patients indicated that hospital contamination was no more a source of infection than the non-hospital environment. Early incision and drainage are imperative. The authors consider that infants who show signs of upper respiratory tract infection should be removed from the breast, isolated and treated with antibiotic drugs. Infants of mothers who develop breast infections should be placed in the isolation nursery and given antibiotic drugs. The ultimate though drastic measure for prevention of breast infection would be to eliminate all breast feeding during periods of increased breast infection. Breast feeding could be resumed when the infection has been curtailed.

Management of Abruptio Placentæ.

L. L. HESTER AND J. SALLEY (*Am. J. Obst. & Gynec.*, December, 1957) present the result of one hundred cases of premature separation of the placenta treated by a consistent regime. These cases occurred in a series of 7434 deliveries, an incidence of 1·3%, or one case in every 76 deliveries. Their routine included a measurement of the height of the fundus and the girth of the abdomen so that any increase in the size of the uterus from concealed hæmorrhage may be detected. Venous blood was obtained for an immediate complete blood count and estimation of the volume of packed cells, and the patient's blood was typed and cross-matched for at least 1500 millilitres of whole blood. Clotting time and clot observation tests were done immediately and repeated hourly until the patient was delivered, or for as long as might be indicated. Blood fibrinogen determination was preferred if available. A transfusion of glucose in water was started so that immediate blood transfusion could be given if indicated. Oxygen was administered by intranasal catheter and the blood pressure checked every five to 15 minutes. In mild cases bed rest and observation was practised, except in the case of patients near term with a ripe cervix when the membranes were ruptured artificially. Blood replacement was given only if the bleeding was excessive. Severe cases were treated with oxygen, blood replacement and artificial rupture of the membranes, and in certain selected cases a "Pitocin" drip was commenced. In this series Cæsarean section was done in three cases, in one because of uncontrolled hæmorrhage, in another because of fœtal distress, and in the third because of suspected rupture of the uterus. Among 102 infants delivered in the series, the perinatal mortality rate was 68%; however, in 50% of the patients no fœtal heart sounds were audible on admission. Most of the neonatal deaths were associated with prematurity, the incidence of which was 58%. The authors consider that with earlier diagnosis there should be a higher incidence of Cæsarean section for fœtal salvage. Twelve cases of postpartum hæmorrhage and nine cases of puerperal morbidity occurred. There were four maternal deaths, two from cerebrovascular accidents and two from renal failure. Severe hypofibrinogenæmia was diagnosed in three cases and was treated effectively with fibrinogen.

SURGERY.

Post-Gastrectomy Steatorrhœa.

W. SHINGLETON *et alii* (*Surgery*, July, 1957) have studied post-gastrectomy steatorrhœa by using triolein and oleic acid labelled with radioactive iodine. They point out that evidence from many sources indicates that following the removal of two-thirds to three-quarters of the stomach by a Billroth II operation, many patients have a mild to moderate degree of steatorrhœa. Following an I^{131} triolein test meal, values of blood radioactivity were significantly lower and fæcal radioactivity higher in a group of such patients as compared with control values. In contrast, the blood radioactivity and radioactive content of the stool reached normal values in two-thirds of this group following an I^{131} oleic acid test meal. The radioactive content of the stool following an I^{131} triolein test meal was significantly lower in patients who had had a Billroth I operation as compared with those who had had a Billroth II type gastrectomy. Dietary history showed no significant difference in caloric intake in that group of patients who were under ideal weight as compared with those whose weight was ideal or above. The addition of bile salts and pancreatic extract to the I^{131} labelled triolein test meal resulted in a striking increase in the blood level of radioactivity in four out of five patients studied.

Coarctation of the Aorta.

E. SMELOFF, S. BAUERFELD AND E. KENT (*Ann. Surg.*, September, 1957) present a series of 60 infants and children with coarctation of the aorta. Twenty-three children were aged from three to 14 years when first seen. Eight of these were operated on with excellent results, and there was no death in this group. Thirty-seven infants were less than 19 months old when first seen, and of these 17 died, a mortality of 46%. Of these 17, six underwent operation and 11 did not. Twelve had preductal coarctations. Four of these underwent operation. Out of 19 with right ventricular hypertrophy, 13 died in infancy and four had been followed for less than six months. All patients selected for operations had been in congestive failure and showed cardiac enlargement, weak to absent femoral pulses, and significant blood pressure differences between the arms and legs.

Aneurysms of the Sinus of Valsalva.

C. LILLEHEI, P. STANLEY AND R. VARCO (*Ann. Surg.*, September, 1957) describe the clinical course of three patients with ruptured aneurysm of the sinus of Valsalva. These three patients illustrate the dramatic onset of disability in cases of ruptured aneurysm of the aortic sinuses. All of these three had severe cardiac impairment, but since operation they have been leading normal lives. The authors made the correct diagnosis pre-operatively. At operation they used total cardio-pulmonary bypass with the bubble diffusion oxygenator and pump, together with retrograde coronary perfusion of potassium citrate to cause cardioplegia. This allowed them to operate on the aneurysms and repair them successfully under direct vision.

Carcinoma of the Lung.

F. COLLIER *et alii* (*Ann. Surg.*, September, 1957) review a series of 600 patients with carcinoma of the lung, of whom 226 were subjected to pulmonary resection, with an operative mortality of 7%. A complete follow-up of 100 patients subjected to pulmonary resection more than five years ago showed a five-year survival rate of 25%, or 27% of those who survived the operation. The presence or absence of blood vessel invasion as seen microscopically in the surgical specimen bore a close relationship to length of survival. Six *per centum* of patients with blood vessel invasion and 75% of those without it had survived for five years. The authors found that in the presence of blood vessel invasion the five-year survival rate was not significantly influenced by the presence or absence of lymph node invasion. In the absence of both blood vessel invasion and lymph node invasion the five-year survival rate was 83%.

Relapsing Pancreatitis.

J. THOMPSON, J. DERRICK AND J. HOWARD (*Surgery*, November, 1957) state that among patients with relapsing pancreatitis recurrent attacks occur in 72% of alcoholic patients; these also have a considerably increased incidence of pancreatic calcification and cyst formation. Those patients with gall-stones tend to have fewer recurrences and they are usually relieved by the removal of the biliary stones. The rate of relapse prior to correction of the biliary tract disease is considerable, but not as marked as in the alcoholic. Removal of the normal gall-bladder does not alter the rate of recurrence. Pancreatitis is therefore a recurrent disease which has a very poor prognosis in the alcoholic patient.

Resection for Traumatic Pancreatitis.

D. W. HANNON AND J. S. SPRAFKA (*Ann. Surg.*, July, 1957) report four cases of rupture of the pancreas due to blunt trauma of the abdomen. Resection of the pancreas distal to the rupture was done in three cases. The quick and complete recovery in the three cases following resection of 20% to 80% of the pancreas suggests that resection is a better treatment than simple drainage. None of these patients had pancreatic insufficiency following this operation.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on October 22, 1957; DR. W. L. CALOV, honorary consultant physician, was in the chair. The principal speaker was DR. J. W. LANCE, honorary assistant physician.

Clinical History.

The subject of the discussion was a man, aged 69 years, who had been well until four months before his admission to hospital, when he had an attack of "virus pneumonia", which left him with a slightly productive cough. When he got out of bed he found that he could not walk, owing to weakness of the legs, especially the left. When he rose from a reclining position he fell to the left, but did not feel giddy. He also complained about his vision. There were no further symptoms other than nocturnal frequency of micturition. He had no headaches, fits or parasthesiæ. Bowel and bladder function was otherwise normal. He had lost no weight. At the age of 29 years he had undergone a right nephrectomy for possible hydatid disease. He said that 10 years previously he had had an accident, resulting in deafness in the left ear. He did not smoke or take alcohol. His parents had lived to old age, and died from irrelevant causes.

On examination of the patient, he was a slightly deaf, thin old man, with normal speech and mentality. The left ear was deaf and the other cranial nerves were normal. The fundi, pupils and pupillary reflexes were normal. There was no nystagmus. There was weakness of the lower limbs, particularly the left. Power of the upper limbs was normal. The finger-nose and heel-knee tests on the left side showed past-pointing. The deep reflexes were equal and brisk, and there was increased tone in the lower limbs. Sensation was normal. The blood pressure was 160/100 millimetres of mercury, and the pulse was regular, the rate being 70 per minute. There was no abnormality in the other systems or in the urine.

A diagnosis of paraplegia was made. X-ray films of the chest, skull and lumbar part of the spine and a Wassermann test of the blood yielded negative findings. There was slight leucocytosis and no anæmia. The first lumbar puncture yielded blood-stained fluid containing 900 erythrocytes per cubic millimetre and occasional leucocytes; the protein content was 140 milligrammes per 100 millilitres, the chloride content 710 milligrammes per 100 millilitres, and the glucose content 80 milligrammes per 100 millilitres. The result of the colloidal gold test was "0001222100". The electro-encephalogram showed no abnormality. Ten days later, examination of the patient revealed nystagmoid movements on looking to the side, a marked intention tremor in the left upper limb, inability to walk unaided and a tendency to fall. His gait was shuffling. The superficial reflexes were normal and the left knee jerk was increased. The other signs were similar to those noted above. An X-ray examination of the cervical part of the spine now showed well-marked degenerative changes in the disk between the fifth and sixth cervical vertebræ. The disk was thin, and there were reactive changes in the adjacent bones. A diagnosis of cervical spondylitis was considered and physiotherapy was commenced. He improved initially, but 36 days later had made no further progress and was unable to walk unaided. Again nystagmus on looking to the side became more marked, and further X-ray examination of the cervical part of the spine showed no evidence of any lesion. The second lumbar puncture yielded clear fluid under a pressure of 170 millimetres of cerebro-spinal fluid. The Queckenstedt test produced a positive result, the Wassermann test a negative result. The protein content of the cerebro-spinal fluid was 300 milligrammes, the chloride content 750 milligrammes and the glucose content 90 milligrammes, all per 100 millilitres. The result of the colloidal gold test was "4322222100"; 15 to 20 erythrocytes per cubic millimetre were present. Two months after his admission to hospital, the right pupil was larger than the left. Nystagmus was present on looking to either side, the rapid component being to that side. Dysdiadokokinesis and past-pointing were present on the left side. The pupillary reflexes and sensation were normal. The deep reflexes were brisk.

A consultant's findings were as follows: mild left hemiparesis involving the face, with some ataxia of the limbs on the left side and dysdiadokokinesis to a mild extent in the left upper limb; there was no clinical suggestion of raised intracranial pressure; the disks were flat and the plantar reflexes flexor. The consultant requested another X-ray examination of the skull and chest, which gave negative findings, and lumbar puncture, which yielded clear fluid under a pressure of 110 millimetres of cerebro-spinal fluid; the response to the Queckenstedt test was positive and that to the Wassermann test negative; the colloidal gold curve was "43222221000". The protein content of the fluid was 250 milligrammes, the chloride content 720 milligrammes and the glucose content 77 milligrammes, all per 100 millilitres. The response to the Casoni test was negative. This consultant now found absent corneal sensation on the left side, left motor and sensory trigeminal loss and left pyramidal tract and cerebellar signs.

Clinical Discussion.

DR. W. L. CALOV: I call on Dr. Lance to present the discussion on this case.

DR. J. W. LANCE: In essence, this is the story of a 69-year-old man who developed a lesion of the brain-stem following a respiratory illness.

There is apparently nothing of interest in the family history. I like the phrase about his parents dying of "irrelevant causes". It is like the patient who, when asked about the deaths in the family, replies: "I don't quite know, doctor, but I'm sure it was nothing serious."

The past history includes a right nephrectomy for "possible hydatid disease". We are informed later that the response to the Casoni test is negative, so I doubt whether a hydatid is going to rear its ugly scolex in this case. The patient has nocturia and a mildly elevated blood pressure, so the function of his one remaining kidney may possibly be suspect. At the age of 69, several nocturnal visits may reasonably be put down to a large prostate, so we can relegate renal function to the background. The other gleam of light that has shone over his preceding 69 years of life is of more importance—deafness in the left ear ten years ago "resulting from an accident". If the accident resulted in a fracture of the petrous temporal bone or a penetrating wound of the middle ear, this may be an acceptable statement. It is quite possible that the onset of deafness in the left ear was gradual, and noticed for the first time shortly after an accident and attributed to it. People are remarkably tolerant of a unilateral deafness, and the symptom has to be sought, as it is rarely volunteered. It should be a routine question at every neurological examination: "Are you deaf in either ear?"

Four months before admission, the patient suffered a chest infection, and this did not completely resolve, as he remained with cough and sputum. Even in a non-smoker, this immediately sounds the familiar warning of bronchogenic carcinoma, which can affect the nervous system directly by metastases, or indirectly by interference with metabolism, giving rise to neuropathy or cerebellar degeneration. This possibility is not completely negated by the later chest X-ray film, which was said to be normal. It would also be worth while inquiring about the treatment that this patient was given for his chest infection. Streptomycin can affect the vestibular nerve easily in old people, even when renal function is within normal limits. As little as one gramme per day for 12 days can permanently impair vestibular function in the elderly. In renal failure, the amount required is much less—as little as three grammes. This vestibular dysfunction causes ataxia as described in this patient, but not the other signs which developed subsequently.

When our patient arose from his bed after his chest infection, both legs were said to be weak and he fell to the left. Falling to one side may be a symptom of a cerebellar or vestibular lesion. From the examination at this time, he appears to have had cerebellar signs on the left side (lack of coordination in finger-nose and heel-shin tests on the left), and also he had weakness and hypertonia of both lower limbs, particularly on the left. The reflexes were brisk and equal and the plantar responses presumably flexor, as this is noted later. Sensation (which includes position sense) was reported as normal. The cranial nerves were normal, apart from the sinister unilateral deafness. It is important to know whether this was of nerve or conduction type to tuning-fork tests. The condition was considered to be one of paraplegia, or more correctly paraparesis, and the lumbar spine was X-rayed. In view of the evidence of cerebellar dysfunction, it is not surprising to find this X-ray picture normal. At this stage we are thinking of a lesion affecting the brain-stem and cerebellum, and in a man of this age a vascular lesion should be considered. However, arterial thrombosis which affects the cerebellum, such

as the posterior inferior cerebellar artery syndrome, does not involve the pyramidal tracts, and we know this man had weakness in both lower limbs, and there is usually evidence of other nuclei (fifth, sixth, occasionally seventh, *nucleus ambiguus*, and vestibular nucleus) and sensory and sympathetic tracts being involved. The condition is unlikely to be a pure cerebellar atrophy, as motor weakness is present. Already our attention is focused on the possibility of a space-occupying lesion in the posterior fossa. An X-ray picture of the skull was reported normal; it would be of interest to review this film and study the apex of the petrous temporal bone and the internal auditory meatus. I have all the X-ray films, and with your permission, Mr. Chairman, would like to point out some features in them after the discussion. The electroencephalogram was also reported as normal.

Lumbar puncture produced blood-stained fluid, probably traumatic, as no headache has been noted. We have no reason to believe he had subarachnoid hæmorrhage. Protein in the cerebro-spinal fluid was raised (140 milligrammes per 100 millilitres), and the colloidal gold test showed a non-specific abnormality, which could be explained by the contamination of the fluid with blood. If the red cells were genuine constituents, angioma or aneurysm would enter the picture. The raised protein lends support to our thoughts of a posterior fossa tumour.

The patient's condition progressed steadily, the left-sided cerebellar signs becoming more prominent and the left knee jerk becoming brisker than the right. X-ray examination of the cervical spine showed thinning of the disk between the fifth and sixth cervical vertebræ, and a diagnosis of cervical spondylosis was considered. This could not possibly explain the cerebellar signs, and as some degree of disk degeneration is common at this age, it is not of great significance.

After 36 days he was unable to walk unaided, and definite nystagmus to the left was noted. It is a little confusing to see that the cervical spine was again radiologically examined and no lesion found. We are not really concerned with the cervical spine at the moment; but the films were reviewed for interest, and both X rays showed degeneration of the disk between the fifth and sixth cervical vertebræ. The second film was so blurred as to render diagnosis difficult.

A further lumbar puncture disclosed that the cerebro-spinal fluid protein had risen to 300 milligrammes per 100 millilitres, and that there was a first-zone Lange curve tending toward the paretic type. A paretic Lange curve occurs in the following disorders: (i) General paralysis of the insane, meningo-vascular syphilis and rarely tabes. In this patient the Wassermann reaction was negative in blood and cerebro-spinal fluid, so we can ignore the possibility of syphilis. (ii) Demyelinating diseases—disseminated sclerosis and Schilder's disease. These are the classical disorders in which a paretic curve and negative Wassermann response are found. These, however, belong to an earlier age group. The steadily progressive course is against the former, and the absence of cerebral symptoms against the latter. (iii) Subacute inclusion body encephalitis. This is a progressive disease of children, with death after a short course. The oldest recorded case was about 20 years. (iv) *Polyarteritis nodosa*. The nervous system is usually involved as a symmetrical polyneuritis, but the central nervous system is affected in approximately 8% of cases. The symptoms are varied, and can simulate most diseases. (v) Cerebral tumour. An abnormal Lange curve depends on the amount of globulin in the cerebro-spinal fluid and its relationship to other proteins. If the cerebro-spinal fluid protein is above 100 milligrammes per 100 millilitres, the Lange curve may be abnormal;. Merritt quotes 25% of cerebral tumours as showing an abnormal Lange curve, and this is of the first-zone type in 5%. (vi) Brain-stem encephalitis. This is a recently described condition resembling, in a way, infective polyneuritis, and is mentioned only for completeness.

Two months after his admission to hospital the right pupil was found to be larger than the left. This may indicate an early left Horner's syndrome consistent with a left brain-stem lesion. A consultant now confirmed the left cerebellar signs, and found a mild left hemiparesis involving the face, with bilateral flexor plantar responses. No mention is made of weakness of the right lower limb at this stage. There was no suggestion of raised intracranial pressure. X-ray films of the skull and the chest were again negative. Lumbar puncture showed little change in the cerebro-spinal fluid picture.

After this, the clinical findings were clarified by the demonstration of left motor and sensory trigeminal loss with an absent left corneal reflex. This places the site of the lesion definitely in the region of the lower part of the pons. Taken in conjunction with the left-sided cerebellar signs and with the left-sided deafness, it suggests a lesion of the left cerebello-pontine angle. Why, then, have we a left hemiparesis? One could expect the right side to be involved, as the pyramidal tracts decussate below the lesion. A space-occupying lesion can force the brain-stem across so as to compress the opposite cerebral peduncle against the tentorium. If this is the explanation in this case, it is strange that there is not raised intracranial pressure, as compression of the aqueduct might be anticipated.

What investigations would help us to reach a conclusion in this case? First, further investigation of the deafness by clinical tuning-fork tests and audiogram. If the deafness is of the nerve type and loudness recruitment is absent, an eighth nerve tumour is a likely diagnosis. If caloric responses are absent on the left side, indicating loss of vestibular function, an eighth nerve tumour is almost a certainty. Air studies. If a cerebello-pontine angle lesion is present, distortion of the fourth ventricle and aqueduct occurs, and air in the pontine cistern may actually outline the lesion. If the above investigations are negative, angiography would be advisable to exclude a brain-stem angioma or aneurysm.

In summary, I feel that this patient had a left cerebro-pontine angle lesion, most probably eighth nerve tumour. That is an acoustic neurinoma or Schwannoma. This implies that the patient's unilateral deafness, which dated back at least 10 years, was really his initial symptom. A fifth nerve neuroma may also occur in this site, but is less common, and in our case fifth nerve involvement was late, so this is unlikely. Meningioma and aneurysm may also be found in this site; but the high cerebro-spinal fluid protein and clinical story are in favour of acoustic neuroma. In this event, the paretic-type Lange curve must be caused by the increase in protein. Other possibilities which we cannot exclude on evidence given are: (i) primary glioma of the brain-stem, which may be insidious; (ii) secondary neoplasm—perhaps from a carcinoma of the lung, related to his previous illness; (iii) angioma of the brain-stem, which may remain quiescent until middle or late life before declaring itself; (iv) syringobulbia, or other degenerative lesions. However, I feel as strongly as I ever can in such conferences that the patient has an eighth nerve neurilemmoma.

In conclusion, I might hazard a guess that the reason why the mode of death is not mentioned in the protocol is that a craniotomy was performed, and the patient was subsequently transferred to the care of the pathologist.

DR. CALOV: Thank you, Dr. Lance, for that interesting and lucid discussion.

DR. W. EVANS: My first thought on reading the protocol was that the patient had a cerebello-pontine angle tumour. But I had difficulty in explaining the fact of cerebellar signs being present on the same side as the pyramidal tract signs. I thought that in such a case we should have more evidence of increased intracranial pressure, and even signs of choked disk. Thus I questioned whether there may not be a multiple lesion. A lesion involving the cervical cord just below the decussation of the pyramids would involve the pyramidal tracts, and cerebellar signs could be as prominent in that region as they would be with a lesion higher up. However, this had to be considered, particularly as there was quite definite evidence of cervical block, as shown by the amount of protein and the positive Queckenstedt. The possibility of a multiple lesion must arise. Multiple sclerosis is almost excluded by the patient's age and by the other symptoms. Other lesions of multiple character that attracted me were multiple malignancy, possibly angiomata in that region. This could give rise to red cells in the cerebro-spinal fluid. The symptoms would have been more pronounced. Another lesion that seems to me quite a possibility is torulosis. Torular infection of the cord spreading up to the brain could account for these symptoms. I have seen two cases of torular infection recently affecting the brain with small abscesses in the brain, and I think this diagnosis has to be considered, even though the cerebro-spinal fluid findings do not indicate this. However, it could explain the apparent multiple lesions which we have to seek.

DR. H. S. H. WARDLAW: I suggest that the difference between the first and subsequent Lange curves may be due to the presence of blood, and the lower protein level in the first specimen of cerebro-spinal fluid. Dr. Lance has pointed out that abnormal Lange curves are due essentially to the presence of raised proportions of globulins. The curves are affected by the levels as well as the proportions of protein. With higher values of total protein, greater diluting may

be required to remove the protective action of albumin, causing the maximum of the curve to move towards the third zone.

Dr. W. L. Calov: Like Dr. Evans, I have been somewhat puzzled by the signs being all on the one side. As Dr. Lance has already pointed out, if the lesion at the cerebello-pontine angle was large enough to push the brain-stem to one side, one would think that there should have been some evidence of raised intracranial pressure. Therefore, although I think Dr. Lance has made out an impressive case for his diagnosis, I feel some slight reservation about it and prefer to sit on the fence. I call on Dr. Lance to review the X-ray films to which he has had access.

Dr. Lance: First of all, in reference to the first spinal X ray, which showed a degeneration of the fifth and sixth cervical vertebræ, and the second X ray, which was reported as normal. These films show, however, that appearances similar to the first are also present in the second X ray, but, as already mentioned, this is a common occurrence in persons of this age, and in my opinion is of no significance in this case.

In the X rays of the skull, however, there is much more significance. First, the appearance of decalcification of the posterior clinoid processes in the lateral view. This is a change that may occur in old people, and is of less significance than a similar finding in young persons, where it could possibly indicate raised intracranial pressure. The antero-posterior views show that in looking through the orbits, the apex of the petrous temporal bone is distinctly seen on the right side. On the left side, however, there is an area of rarefaction in the petrous temporal bone, such as is seen in a space-occupying lesions in its vicinity. In another antero-posterior view, the internal auditory meatus is easily seen on the right side. On the left side, although the interpretation is slightly equivocal, it appears that there could be an expansion of the internal auditory meatus. This could be verified by a Townes and Stenver's view of the area. However, it cannot be denied that there is some rarefaction of the petrous temporal bone on the left side. This supports the diagnosis of a lesion in this area, and I am in favour of its being an eighth nerve tumour.

Dr. Calov: I call on Dr. Hirst to reveal the findings.

Dr. E. Hirst: At this stage the history in the protocol has led us to the diagnosis of probable cerebello-pontine angle tumour. The remainder of the history is to follow. The patient refused operation, and was discharged. Seven weeks later he had an epileptiform fit and was readmitted to hospital, where he still refused operation, and remained under observation until death. This gives us an opportunity to study the natural course of his disease.

On readmission, he was now conscious of weakness on the left, and said he could walk only if supported on the left side. He had trouble in picking up objects. If he tried to pick up an object it would float off to the left. There was numbness around the mouth and towards the left eyelid. He had lost two stone in weight. On examination, he appeared to have difficulty in fixing his gaze. His pupils were dilated and reflexes sluggish. He had gross ataxia and Rhombergism. His speech was slurred, and there was nystagmus on looking to either side. Ptosis was doubtful. Tactile sensation was impaired on both sides of the face. A rebound phenomenon was elicited on the left side. A fourth lumbar puncture revealed findings similar to those previously obtained. The protein was 100 milligrammes per 100 millilitres and the Lange curve of the paretic type. Physiotherapy was continued all this time, and three months after this second admission he had diplopia, left third and sixth nerve palsy, and an upper motor lesion of the seventh nerve. He now developed a bed sore and became incontinent of urine. He began to lose interest in his surroundings, his speech became slurred and disjointed, and he would ask when his treatment was going to commence. Exactly a year after his admission, and 16 months from the onset of his first symptoms (other than the deafness), he became pyrexial and comatose and died. The notes do not contain information on the type of deafness or on caloric tests, possibly not pursued in view of his attitude to treatment.

At post-mortem examination, he was a thin man with prominent eyes. There was no abnormality suggestive of neurofibromatosis, such as café-au-lait patches or tumefactions of the peripheral nerves or of the skin. The right kidney was absent, and the left was grossly normal. The prostate was, as Dr. Lance suggested, slightly enlarged. The significant changes were in the brain and lungs.

The convolutions of the brain were flattened, and there was marked dilatation of the ventricles, indicating internal hydrocephalus and no doubt raised intracranial pressure, which was, however, not confirmed clinically at any time. Situated at the left cerebello-pontine angle there was a roughly ovoid new growth approximately three centimetres in maximum diameter and causing marked displacement of the pons and medulla to the right. There was sufficient pressure to produce hydrocephalus in the absence of clinical signs of raised intracranial pressure. This was noted by Dr. Evans and Dr. Calov as well as by Dr. Lance. The cut surface of the growth showed areas of hæmorrhage and of fairly deep yellow discoloration, as well as cysts set in the general background of semi-translucent tissue. These gross appearances are due to areas of hæmorrhage and large dilated vascular spaces, and to accumulation of lipids in histiocytes, which is very characteristic of acoustic neuromas. Areas of hyaline degeneration account for the translucent appearance of parts of the growth, and the accumulation of protein-rich fluid in the cystic spaces is also characteristic. It is possible that the escape of this fluid into the cerebro-spinal fluid accounts for the high protein notable in this growth. The characteristic histological appearance of palisading of the Schwann cells is also seen in other parts of the growth. The growth was adherent to the petrous temporal bone adjacent to it, and this corresponds to the radiological findings that Dr. Lance emphasized. The main changes in the lungs were seen in the right upper lobe, which on gross inspection had patchy consolidation suggestive of bronchopneumonia, and this was confirmed on section.

In summary our findings were: left acoustic neurilemmoma with bony erosion and right suppurative bronchopneumonia.

Dr. Lance: I should like to point out again the importance of the unilateral deafness present in this patient. In any patient with unilateral deafness, it should be determined immediately if it is nerve deafness. If it is, caloric responses, audiometry and loudness recruitment tests should be performed. Loudness recruitment is absent in eighth nerve tumour, but is present in Ménière's disease and in labyrinthine degeneration. If loudness recruitment is absent and the audiogram and clinical tests show nerve deafness and caloric responses are diminished or absent on that side, then it is guineas to gooseberries that it is an eighth nerve tumour. Good views of the internal auditory meatus, including Stenver's view of the petrous temporal bone, will confirm the diagnosis, which will be rendered certain by air studies.

Dr. H. M. Whyte: Could you indicate the results of surgery in these cases?

Dr. Lance: This is an important question, and the results of surgery are, unfortunately, not as good as we should like. The presence of an eighth nerve tumour is not an absolute indication for operation. Even in expert hands, facial palsy is not infrequent except in early cases. Other complications are hemiparesis or even death from interference with brain-stem functions.

If the diagnosis is made very early, at the stage when only the eighth nerve is involved, operation is indicated, provided the general health of the patient is good. The growth in these cases can usually be removed and the neighbouring structures spared. At later stages when the brain-stem is involved, operation is extremely hazardous and may be contraindicated. At a still later stage, when raised intracranial pressure supervenes, or such gross signs that the patient is becoming disabled so that anything is better than his present condition, then again operation is justified though the prognosis is grave. It is ideal to find the growth at an early stage—that is, at the stage where deafness is the only symptom—when the outlook after operation is very good.

Dr. B. P. Billington: I should like to ask if an eighth nerve tumour is a relatively frequent incidental finding at autopsy.

Dr. Hirst: Our recent records show four such tumours during the last year. As in this case, the patients came to operation or to autopsy, and none of the growths were incidental.

Dr. A. A. Palmer: I can remember seeing one acoustic neurinoma as an incidental finding, but I think that bilateral eighth nerve tumours in cases of neurofibromatosis may be found, because in neurofibromatosis the incidence is higher than in the rest of the population.

Dr. Calov: I would like to ask Dr. Lance for a likely explanation of the absence of raised intracranial pressure on clinical examination.

DR. LANCE: I found some difficulty in considering this point. I presume that there was sufficient flow along the aqueduct to maintain free drainage of the ventricular system, even though the peduncles were compressed. They could have been compressed ventral to the aqueduct and spared the aqueduct. I think that must be the solution, since compression of the aqueduct itself usually brings about the final *dénouement*.

DR. WHYTE: Is there any suggestion of why the tumours occur at that site, and why they choose the auditory nerve?

DR. LANCE: Schwannomas can occur on any nerve. I do not know why there is frequent involvement of the eighth nerve, but the fifth nerve may sometimes be involved. Also the ninth nerve and the peripheral nerves and spinal nerve roots. It used to be said that the first cervical root was an exception, but we recently had one such tumour on the first cervical nerve root.

PROFESSOR W. K. INGLIS: This is an interesting subject. The illustrations of the histology of that tumour were very clearly cut, and they showed the "palisading" particularly well. That "palisading" is characteristic of the so-called Schwannoma or neurilemmoma, which arises from the sheath cells surrounding myelinated nerves of peripheral distribution. In ordinary neurofibromatosis the tumours are quite different (referring, of course, to peripheral nerves), and it has been suggested, in particular by Del Rio Hortega, that they arise from different cells—namely, sheath cells surrounding non-myelinated nerves. Both varieties of sheath cells are of neural crest origin. The point is, however, that in peripheral distribution there is a clear-cut histological distinction between neurofibroma (or, as Del Rio Hortega calls it, the lemmocytoma) on the one hand, and the Schwannoma (or neurilemmoma with "palisading") on the other.

Central neurofibromatosis occurs. The curious thing about central neurofibromatosis is that bilateral acoustic nerve tumours are met with, and they have the curious structure, with "palisading", that you have been shown. Central neurofibromatosis, however, very seldom has peripheral distribution of lesions as well. There was one case of cerebral neurofibromatosis described recently in which a single peripheral lesion had a Schwannomatous appearance. Shapland and Greenfield described a case of central neurofibromatosis in which there was a single peripheral tumour thought to be a neurofibroma. But there is something particularly odd about these cases of central neurofibromatosis and acoustic nerve tumours. Why they should have this structure, and why they should pick out those two nerve trunks, I do not know; the subject has led to considerable discussion, and it is still open to question.

DR. CALOV: I would like to congratulate Dr. Lance and thank all those participating in this conference.

Diagnosis.

Acoustic nerve tumour or neurilemmoma.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on October 16, 1957, at St. Vincent's Hospital, Melbourne. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital.

Compression Treatment of Fractures.

MR. T. KING showed a cinematographic film illustrating the use of compression in the treatment of ununited fractures of the shafts of the long bones. In 13 of the ununited fractures sinuses were present, and 12 united. Griefensteiner's method of obtaining compression by two Kirschner wires stretched in a large stirrup was used. Bone union was obtained in 42 out of 50 patients. Mr. King said that compression did not appear to accelerate the speed of bone union, but it was valuable when sinuses were present, especially at the lower end of the humerus and the tibia, as a method of immobilization that "took up" the gap between the two bone fragments at the fracture site caused by infective osteitis especially in cancellous bones.

Cervical Sympathectomy for Internal Carotid Thrombosis.

MR. F. MORGAN and MR. K. HENDERSON showed a man, a retired civil servant, aged 60 years, who had presented in April, 1957, with a history of (i) intermittent claudication for eight years, with exercise tolerance of 100 yards, (ii) transient attacks of right-sided paralysis with aphasia, mostly at night, since 1954 (occasionally the left side was affected), and (iii) temporary paralysis of the right leg, occurring nightly at 3 to 4 a.m. for the past week, with gradual return of normal function to the limb after one hour. On examination of the patient, he was arteriosclerotic and hypertensive (blood pressure 210/100 millimetres of mercury). No arterial pulsation was felt in the legs except in the femoral arteries, and the left carotid artery was impalpable. There were no definite neurological signs, but the plantar reflexes were equivocal. X-ray films of the skull and legs were normal, and an electrocardiographic examination made by Dr. J. Horan was reported to reveal no abnormality. The impression was of thrombosis of the arteries of the lower limbs and occlusion of the left internal carotid artery. The left superior cervical ganglion was respected on June 14, since when he had had no further weakness of the right limbs. However, his exercise tolerance was now only 50 yards, and he was requesting lumbar sympathectomy for relief of the intermittent claudication.

Trans-Sphenoidal Operation for Pituitary Adenoma.

Mr. Morgan and Mr. Henderson next showed an engineer, aged 40 years, who had presented in August, 1955, with symptoms of bilateral fronto-temporal headaches for 12 years and failing vision for two years, the left eye being first affected. There had also been "queer turns" for eighteen months, in which he sank to the ground almost unconscious; they had the characters of minor epilepsy. He was also subject to tiredness and fits of depression. On examination of the patient his skin was firm and smooth, but his hair was normal in texture and distribution. He shaved daily. The optic fundi were normal. Visual acuity was 5/5 with Snellen type and 10 with Jaeger type in the right eye, and 5/18 (Snellen) and 18 (Jaeger) in the left eye. The fields had a rather bizarre pattern with a tendency to right homonymous hemianopia, especially in the upper quadrants. In conjunction with the arteriographic finding of elevation of the left carotid artery, those findings indicated extension of a pituitary adenoma outside the *sella turcica* across the floor of the middle fossa, with involvement of the left optic tract. A trans-sphenoidal approach to the pituitary fossa was made on August 26, 1955, and a subtotal removal of the tumour effected by suction. Visual acuity improved to 1 (Jaeger) and 5/3 (Snellen) in the right eye and 4 (Jaeger) and 5/18 (Snellen) in the left. However, his vision deteriorated over the next few months, and with it his mental state. In January, 1956, he had left optic atrophy and incomplete right homonymous hemianopia, and the visual acuity was 6/5 (Snellen) and 1 (Jaeger) in the right eye, but only 6/50 (Snellen) and 20 (Jaeger) in the left eye. X-ray films now showed enlargement of the sella with destruction of the *dorsum sellæ* and elevation of the anterior clinoid processes. The cerebro-spinal fluid contained 40 milligrammes of protein per 100 millilitres (Pandy "+") and three white blood cells per cubic millimetre. A left temporal osteoplastic craniotomy was performed on February 27, 1956, and a further subtotal removal was made of a large adenoma with retro-chiasmal extensions and extradural extension beneath the left optic nerve and carotid artery. It had also filled the cavernous sinus. Recovery from the operation was very satisfactory. When the patient was last seen on March 21, 1957, visual acuity in the left eye was 5/9 (Snellen) and 8 (Jaeger), and the fundus appeared almost normal. There was slight third nerve paresis, sustained at operation, but that was decreasing.

Operative Treatment of Hyperostosing Meningioma.

The next patient shown by Mr. Morgan and Mr. Henderson was a male civil servant, aged 55 years, who had presented in January, 1955. He complained of (i) a lump on the head present since a fall from a bicycle at the age of seven years, (ii) frontal headache, inability to concentrate and difficulty in writing for two and a half years, (iii) sudden onset of weakness of the right arm one year earlier, (iv) weakness and dragging of the legs, especially the right, for six months, and (v) mental irritability for six months. On examination of the patient, he was cooperative, but his memory was poor and his responses were retarded. Papilœdema of the left disk and slight hemiparesis on the right side were present. A discoid hyperostosis was present over the bregma, measuring 3·0 by 2·75 inches. It was tender. X-ray films

of the skull showed hyperostosis of the porous spongy bone about the site of the bregma, and the pineal gland was displaced backwards and slightly to the right. The spenoparietal venous sinuses were enlarged. Air encephalography showed the left lateral ventricle to be displaced downwards and indented from above. Bilateral common carotid arteriography showed displacement of the right anterior cerebral artery to the right and downwards and back. The electroencephalographic findings were within normal limits. The impression was of a hyperostosing meningioma of the falx, and this diagnosis was confirmed at operation on February 17, 1955, at which a bilobed meningioma of the falx was removed and a block resection made of the skull about the bregma. The bone was scraped and levelled, remoulded, boiled and replaced *in situ*. Convalescence was very satisfactory, despite weakness of legs for a few days. The patient had remained well except for a temporary episode of psychotic depression, in which he attempted to cut his throat and had to be certified to a mental hospital. He recovered with treatment and was now back at work.

Leucotomy for Agitated Depression.

Mr. Morgan and Mr. Henderson then showed a married woman, aged 49 years, who since October, 1954, had been dominated by depressive symptoms with delusions of unworthiness and exaggerated ideas of guilt. She felt that she was damned and too wicked for God's mercy. She was convinced that her mind was possessed by the Devil, and was obsessed with impure and sacriligious ideas about God. For some weeks she had given up the practice of her Roman Catholic faith, because of her belief that she was too evil for it. She had had short-lived relief from her symptoms after two courses of electroconvulsive therapy late in 1954, but she soon relapsed, especially after the second. Examination of the patient showed her to be of pyknic build with a florid complexion and acne. She had hypertension (blood pressure 190/110 millimetres of mercury). She talked almost incessantly about her wickedness, and was completely inaccessible to reason or reassurance. Leucotomy (Scoville's orbital undercutting operation) was undertaken on August 11, 1955. She had immediate improvement, with restoration of a normal personality within a few days. She had resumed full control of her household duties and had been completely cured of all symptoms since the operation. In September, 1957, she was readmitted to hospital because of headache and left-sided homonymous hemianopia due to hypertensive vascular disease of the left posterior cerebral artery. The symptoms did not cause any deterioration in her mental state, and cleared up after two weeks in hospital.

Chronic Subdural Hæmatoma.

A further patient shown by Mr. Morgan and Mr. Henderson was an Italian labourer, aged 34 years, who had fallen off a truck and struck his head on a concrete floor four months earlier with five minutes' loss of consciousness. He had begun to suffer from constant bifrontal and bitemporal headache one week after injury; headache awoke him nearly every morning at 3 to 4 o'clock. He had occasional vomiting attacks. Examination of the patient revealed bilateral papilœdema with hæmorrhages and exudates, minimal left facial weakness with reduced superficial abdominal reflexes on the left, a depressed supinator jerk on the left side, a depressed Meyer reflex on the left, and doubtful weakness of the left upper limb. X-ray films of the chest and skull revealed no abnormality. A right carotid angiogram showed a small collection on the right, but the anterior cerebral arteries were displaced to the right, indicating a larger collection on the left side; this was subsequently confirmed by angiography. The pathological finding was chronic subdural hæmatoma. At operation on March 26, 1956, a large chronic subdural hæmatoma was removed from the left side. The post-operative course was uneventful.

Recurrent Pituitary Chromophobe Adenoma.

Mr. Morgan and Mr. Henderson then showed a married woman, aged 29 years, who had presented on November 29, 1954, with the following complaints: (i) progressive failure of vision for one year, with complete blindness for two months; (ii) amenorrhœa for five months; (iii) loss of energy and easy fatigability present for several months; (iv) loss of memory and personality changes present for several months; (v) headaches, frontal and bitemporal, especially occurring in the morning, and often associated with nausea and vomiting. Examination of the patient showed her to be a slightly plethoric woman with no definite evidence of Cushing's syndrome. Mentally she was slow and lethargic, but well orientated. Investigation of the cranial nerves revealed blindness in the right eye with right optic atrophy, left temporal hemianopia and slight left facial paresis. Increased tone and reflexes were found in the left limbs, and the plantar reflexes were flexor in type. A Wassermann reaction was negative. Lumbar puncture produced clear fluid under a pressure of 230 millimetres of water; the fluid contained no cells and 100 milligrammes of protein per 100 millilitres; Pandy's reaction was "++". An X-ray examination of the skull revealed asymmetrical enlargement of the pituitary fossa; the optic canals were normal. An arteriogram showed a displacement of the right syphon and middle cerebral artery backwards. On December 16 right frontal craniotomy was performed. After removal of the tip of the right frontal lobe, subtotal removal of a large tumour arising from the pituitary fossa was carried out. The pathologist's report was: "Well differentiated chromophobe adenoma of the pituitary." After operation the patient was given cortisone replacement therapy, but became very mentally disorientated, this state persisting for several weeks.

She was readmitted to hospital on September 5, 1957. She had remained well after her previous discharge, her menstrual periods recommencing three months later. The patient had become pregnant eight months previous to her second admission to hospital. She gave a history of the sudden onset of impaired vision in the left eye six weeks previously, which had progressed. The right eye had been blind since her last admission. For two weeks she had had occasional difficulty with her speech, with episodic nominal dysphasia. She had suffered from thirst for four weeks, and polyuria was suspected of being present. Examination of the patient showed that the fundus was enlarged to the size of a 36 weeks' gestation. She was alert and cooperative. Investigation of the cranial nerves revealed impaired sensation, blindness of the right eye (the visual acuity of the left eye was 2/36), optic atrophy of the right fundus, and temporal pallor in the left fundus. No abnormality was detected in the peripheral nervous system. An electroencephalographic examination revealed a soft tissue mass projecting above the *sella turcica* and into the right frontal horn; the mass was also deforming the antero-inferior part of the third ventricle. Examination of the cerebro-spinal fluid showed it to contain no cells, and 30 milligrammes of protein per 100 millilitres. Examination of the visual fields revealed altitudinal loss of the lower half of the left field with macular impairment.

A diagnosis was made of a recurrence of pituitary adenoma, and craniotomy was performed on September 12; a recurrent pituitary adenoma was found, and was subtotally removed. After operation the patient was given cortisone, but became mentally disorientated. Further treatment (irradiation) and investigations relating to hypopituitarism were deferred till after parturition, and the patient was transferred to the maternity hospital for Cæsarean section.

Repair of Dural Tear with Fascia Lata Graft.

Mr. Morgan and Mr. Henderson finally showed a male Italian, aged 25 years, who 10 years earlier in Italy had sustained a compound fracture of the skull from a motor-car accident, the laceration being over the right frontal region; he had been discharged from hospital after one month, quite well. Seven years later he had developed meningitis, and had been in hospital for 35 days, after which he was discharged, well. Nine weeks before his admission to St. Vincent's Hospital after a previous upper respiratory tract infection, he had developed meningitis, and had been admitted to a country hospital. On examination of the patient at St. Vincent's Hospital, he was seen to be healthy and well built. There was a linear scar in the right supra-orbital region with underlying thickening of bone. Neurological examination revealed no abnormality. X-ray examination of the skull revealed a fracture involving the inner plate of the right frontal sinus. On September 16 right frontal craniotomy revealed a linear fracture extending from the inner plate of the right frontal sinus across the superior aspect of the ethmoid sinus to the cribriform plate. The dural tear was repaired with a *fascia lata* graft. The post-operative course was uneventful, and the patient was discharged from hospital, well.

Epilepsy.

Dr. J. J. Billings discussed the diagnosis and treatment of epilepsy. He emphasized the recognition of epilepsy as a symptom and not a disease, and showed charts illustrating the organic disorders which might give rise to epilepsy. There were illustrations of the localization of an epileptic focus by the electroencephalogram. X-ray films of a number

of patients were presented, including the following: (i) a patient with a right frontal lobe abscess, the first symptom of which was epilepsy; (ii) a patient with a parietal lobe tumour, who had focal epilepsy beginning in the right hand and spreading over the right side of the body; (iii) a patient with a large arterio-venous malformation complicated by epilepsy and subarachnoid hæmorrhage; (iv) a patient with carcinoma of the lung and secondary carcinoma of the brain, whose presenting symptom was epilepsy of a focal character affecting the left leg and foot. Tables of anticonvulsant drugs were presented, and the management of different types of epilepsy was discussed.

Thyroid Disease.

DR. G. BROSNAN showed 17 patients to illustrate various phases of thyroid diseases.

Two patients were suffering from Hashimoto's disease. Both had been operated on for pressure symptoms on the trachea and œsophagus; they showed at the time of the meeting slight evidence of hypothyroidism, and were under treatment with thyroid extract.

One patient had had a so-called "lateral aberrant thyroid" excised seven years earlier, with a subsequent subtotal thyroidectomy and removal of the lymph glands from the right side of the neck. Histological examination of sections revealed a small papillary carcinoma in the right lobe of the thyroid with secondary deposits in the lymph nodes. The patient was well, and showed no evidence of recurrence.

Another patient illustrated the existence of a true lateral aberrant thyroid. Four years earlier she had had an isolated swelling removed from the right side of the neck, found to consist of thyroid tissue, there being no evidence of malignancy. Soon afterwards a subtotal thyroidectomy was carried out, and a careful search was made for histological evidence of neoplasm; however, none was found. The patient had remained well, and was still being kept under observation to see whether any recurrences appeared. So far she was regarded as having had a true lateral aberrant thyroid.

One patient illustrated the presence of a nodular goitre in the neck, with a fairly large retrosternal extension. Her main symptom was dyspnœa. She was awaiting operation.

One patient had presented with a toxic nodular goitre during pregnancy. She was treated with "Neomercazole" until after a normal confinement, and about four months subsequently underwent subtotal thyroidectomy.

Another patient presented with a smooth, rounded swelling on the posterior third of the tongue, in the mid-line; clinically no thyroid tissue could be palpated in the neck. That was regarded as a lingual thyroid, but as no symptoms were present, and it was most likely the only thyroid tissue the patient had, no surgery was contemplated. The use of radioactive iodine as a diagnostic aid in that case was discussed.

Several patients were shown to illustrate the result of thyroidectomy, special attention being drawn to the value of a good cosmetic as well as clinical result. Dr. Billings said that in that regard the careful suturing of the platysma muscle, followed by the close and accurate opposition of the skin edges without any form of drainage, was recommended.

Pathological Demonstration.

DR. KEVIN O'DAY demonstrated a series of enlarged photomicrographs from the department of pathology. These were sections of various Australian animals, fish, birds, snakes and lizards, and included Pygopus (the limbless lizard), Typhlops (the blind snake), the trout, the Murray turtle, the marsupials, and the monotremes (platypus and echidna).

Ophthalmological Conditions.

DR. KEVIN O'DAY, DR. H. RYAN and DR. E. RYAN then showed a series of patients with advanced retinal vascular changes—two with central arterial obstruction, two with central venous thrombosis, and one case with *retinitis circinata*. Another patient shown was an elderly man, suffering from advanced rheumatoid arthritis, who had an area of *sclero-malacia perforans* in one eye. It was proposed to deal with the lesion by the insertion of a *fascia lata* graft. A patient with myotonic dystrophy was next demonstrated. His cataracts had been extracted, and he was able to read newsprint readily. A man with bilateral papilœdema was then presented; investigation indicated that he had a cerebral tumour near the roof of the fourth ventricle.

Gynæcological Demonstration.

MR. F. HAYDEN and MR. H. G. FURNELL showed pathological specimens and coloured slides of the following gynæcological conditions: long-standing inversion of the uterus treated by vaginal hysterectomy; pregnancy with carcinoma of the cervix; hydradenoma of the vulva; dermoid cyst containing teeth.

The Treatment of Fractures.

MR. W. GAYTON showed several patients to illustrate various methods of treating fractures. These included a patient who had sustained a compound fracture of the tibia with loss of the middle third at the time of the original injury; a fibula bone graft had been used to maintain leg length. A second patient shown had sustained bilateral comminuted fractures of the femur; these had been treated conservatively. A patient who had sustained a central dislocation of the hip was also shown. Mr. Gayton also showed slides illustrating Moore's type of replacement arthroplasty of the hip joint.

MR. J. GRANT showed three patients who had undergone amputations of the upper extremity, and who were wearing efficient arm prostheses.

Lung Infection.

DR. LUKE MURPHY presented a series of case reports, X-ray films and post-mortem specimens illustrating features of various forms of pneumonia.

Three examples of staphylococcal pneumonia in young adults were presented. Two were post-influenzal bronchopneumonia, one fatal. The third was due to an infected embolus from a carbuncle on the arm. The points stressed were the need for early recognition of staphylococcal pneumonia, the value of massive penicillin therapy and the use only of the crystalline form of the antibiotic. In the fatal case (the patient had died some years previously) the penicillin dosage was inadequate by present-day standards. In the other two cases a prompt response resulted from the administration of 1,000,000 units of crystalline penicillin every three hours, even though the infecting strains were reported as insensitive to that antibiotic. A minimum daily dosage of 6,000,000 units was suggested in known cases. Up to 150,000,000 units could be used in severe cases.

The X-ray films of a patient with pneumonia due to Friedländer's bacillus were presented; they showed initial pneumonia in the upper lobe of the right lung, later cavitation and spread to the left lung, with a fatal outcome after four months.

A case was described of pneumonia with effusion due to *Proteus vulgaris* in a young woman suffering from an infected incomplete abortion. It was observed that most cases of pneumonia due to that organism or to coliform bacilli were associated with infected abdominal or pelvic lesions.

A report and X-ray films illustrating pneumonia following metal fume fever were presented. The patient had been exposed to fumes from molten brass in a foundry 12 hours before the onset of his illness. He was unaware of the danger of such fumes. Recovery was complete in a few days. The frequency of milder forms of metal fume fever as an occupational disease in brass foundry workers was noted.

Further cases described illustrated virus pneumonia, uræmic "pneumonia", and a middle-lobe syndrome associated with glandular fever.

Dysphagia.

DR. JOHN HORAN first showed two patients with dysphagia. The first patient was a woman, aged 62 years, who had had that symptom for nine months. The patient said that at first solid foods appeared to stick in the lower part of her chest, and at the time of the meeting in addition pain occurred. She felt nauseated all the time, sometimes dry retched and had vomited once. She had lost three stone in weight. X-ray examination with a barium meal made five months earlier had been reported as showing a "cup and spill" type of stomach, but no other abnormality; a blood count showed that the red cells numbered 2,900,000 per cubic millimetre, the hæmoglobin value was 71% (9·9 grammes *per centum*) and the colour index was 1·2. The patient had been admitted to hospital a week prior to the meeting, and X-ray examination with a barium meal then showed a large neoplasm in the fundus of the stomach and involving the lower end of the œsophagus. That was confirmed at operation. Dr. Horan emphasized the difficulty in diagnosing

carcinoma in that area, since that portion of the stomach was relatively inaccessible to palpation at the bedside and even under the fluoroscopic screen; in addition, films of the opaque meal needed to be taken with the patient prone or in the head-down position. Another feature of that case illustrated a frequent finding in carcinoma of the stomach—a blood count suggesting pernicious anæmia.

The second patient with dysphagia was a man, aged 52 years, who had been admitted to hospital a month earlier. For 25 years he had had *lupus erythematosus* involving the pre-auricular areas of the face and the lobes of the ears, and for which he had received treatment with courses of bismuth and of gold, and more recently had been given "Atebrin". Five months prior to the meeting, after an attack of influenza four weeks previously, he had first noticed difficulty in swallowing solid foods. He had lost approximately two stone in weight, and in the same time had developed weakness in the lower limbs. He was found to have wasting of the glutei, the quadriceps and the muscles of the arm, the deltoid and the spinatus. There was no fibrillation of muscle; the deep reflexes were considerably diminished, and the plantar reflexes were flexor in type. There was no disturbance of sensation. X-ray examination of the chest and radiological examination with a barium bolus revealed no abnormality. Laryngoscopy and œsophagoscopy revealed normal findings also. The blood count was normal and the Wassermann reaction negative. The sedimentation rate was 43 millimetres in one hour, and the blood urea content was 43 milligrammes per 100 millilitres; the serum electrolytes and the plasma proteins were normal. Dr. Horan said that the diagnosis was still in doubt, the possibilities being progressive muscular atrophy, *myasthenia gravis*, muscular dystrophy or a neuropathy or myopathy complicating carcinoma. It was unlikely that the dysphagia was part of progressive muscular atrophy as, in that case, it would be associated with other disturbances in function of the muscles supplied by the bulbar nuclei, occurring early in progressive bulbar paralysis and late in the commoner form beginning in the muscles of the hands. Dr. Horan said that difficulty in swallowing was a common symptom in *myasthenia gravis*, but he pointed out that the dysphagia increased in severity as the day went on, and that many patients suffering from that complaint found that they could eat solid foods early in the day and by evening could swallow only fluids. A diagnosis of muscular dystrophy was considered, but dismissed in the presence of dysphagia, as dysphagia did not occur in this syndrome. Dr. Horan said that Dr. Morgan, who had examined the patient in consultation, raised the question whether he might not be suffering from a neuropathy or myopathy complicating carcinoma, a clinical rarity.

Coarctation of the Aorta.

Dr. Horan then showed a man, aged 31 years, suffering from coarctation of the aorta. Four days before his admission to hospital the patient had awakened with weakness in the left leg. He found that he was unable to support his weight on his left leg and it dragged when he attempted to walk. The weakness diminished steadily in the next four days. When he was admitted to hospital, only slight weakness of the dorsiflexors of the left foot and an increase in the left knee jerk were evident. The apex beat was four inches from the mid-line, and a systolic murmur at the pulmonary area was the only murmur heard. Pulsation could be felt in the thoraco-dorsal arteries in the axillæ, but no pulsation could be seen in the arteries in the region of the scapulæ. Pulsation was not felt in the femoral or the posterior tibial arteries, but a feeble pulse was present in both dorsal arteries of the feet. The blood pressure in the arms was 190/120 millimetres of mercury and in the legs 160/100 millimetres of mercury. X-ray examination of the chest revealed no enlargement of the heart, the aortic knuckle was small, and notching was seen on two of the lower ribs. There was an indentation in the posterior part of the barium-filled œsophagus, considered to be due to an artery arising in an abnormal manner. Dr. Horan said that the patient had coarctation of the aorta of moderate degree. He pointed out that, second only to bicuspid aortic valves, the commonest congenital defect associated with coarctation was an anomaly in the origin of the great vessels, and he considered that the indentation in the œsophagus was due to the right subclavian artery arising from the left side of the arch of the aorta and passing behind the œsophagus. The paralysis of the left leg of upper motor neuron type might have been due to thrombosis from hypertension, but another possible cause was hæmorrhage from a berry aneurysm, another common congenital anomaly found in that condition.

Constrictive Pericarditis.

Dr. Horan finally showed a man with constrictive pericarditis, who had made excellent progress since his operation. Before operation he had had dyspnœa on exertion for four years and ascites for two. The veins in his neck were distended, and when he was sitting up they were visible to within an inch of the lobes of the ears. The blood pressure was 115/80 millimetres of mercury; the venous pressure was 20 centimetres of water. The apex beat was difficult to feel and was four inches from the mid-line; the heart was fibrillating, and there was a third heart sound. There was œdema of the legs to just below the knee with stasis eczema of the lower third. Gross ascites was present; the abdomen was tapped four times before operation, and approximately 50 pints of fluid were removed. Calcification of the pericardium could be seen radiologically. At operation the pericardium was thickened and calcified over both ventricles; it was excised. The ascites disappeared after operation, and the eczema of the legs steadily diminished. The patient had gained almost four stone in weight, and although fibrillation was still present, the dyspnœa had been greatly reduced.

Dermatological Demonstration.

Dr. Denis Clarke discussed a group of unusual skin diseases, and illustrated his remarks by colour photographs. The conditions included pustular psoriasis, cattle ringworm, orf, keratoacanthoma, *molluscum contagiosum*, carcinoma developing from leukoplakia of the tongue, superficial epitheliomatosis, *lupus erythematosus* of the lip and face responding to chloroquine therapy, pyogenic granuloma from *nævus flammeus*, *pityriasis rosea*, Raynaud's disease, trichotillomania, syphilis, pellagra, lymphangioma, *pyoderma gangrenosum*, drug eruptions and *dermatitis artefacta*. There were some illustrations of the technique of dermabrasion.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

A meeting of the Pædiatric Society of Victoria was held on September 11, 1957, at the Royal Children's Hospital, Melbourne.

Hydrocele in Infants and Children.

Dr. R. Fowler, junior, discussed the pathogenesis and treatment of hydrocele in infants and children. He said that the purpose of his paper was to report an investigation into the pathogenesis and treatment of childhood hydroceles which had been carried out during the past two years by the surgical research team at the Royal Children's Hospital. The investigation had been commenced as a prospective study for the deliberate appraisal of the teachings of Dr. Douglas McKay, of Adelaide, who for the past 10 years had practised an original and simple method of treating such hydroceles, based on his theory of their origin. His method ignored altogether the hydrocele sac and struck directly at the source of fluid. The results of the present investigation supported his view as to the origin of such hydroceles and also as to the best method of treating them.

Dr. Fowler said that in considering the possible causes of hydroceles, the choice was limited to three groups: (i) those that were secondary to inflammation or neoplasia of the testis or its adnexæ; (ii) those in which an obvious communication with the peritoneal cavity allowed free passage for peritoneal fluid into the hydrocele sac; (iii) the so-called idiopathic hydroceles, in which no such cause for the fluid accumulation was obvious. Dr. Fowler said that secondary hydroceles were uncommon in childhood, and only one such case had been encountered during the two-year period of the study. Idiopathic hydroceles had a curious age distribution, which had previously excited little comment. Although they were fairly common in childhood and in old age, they were a relative rarity in adolescents or young adults. He believed, therefore, that there was some fundamental difference in the pathogenesis of hydrocele in those different age groups, and the rest of his remarks would be confined to hydroceles in infancy and childhood.

At the outset Dr. Fowler emphasized that a distinction between the communicating and non-communicating hydrocele in childhood could often be made only in retrospect. If objective evidence of reduction of a hydrocele was obtained, then obviously it was of the communicating type.

However, a story of disappearance of the lump or a fluctuation in size, although highly suggestive, was not of itself sufficient evidence to establish the hydrocele as of the communicating type. Most of the children with non-communicating hydrocele gave a history of disappearance or fluctuation in size at some stage in the life history of the hydrocele. With that qualification in mind, his material consisted of 57 children with 61 hydroceles, taken as they came from the waiting list and, in retrospect, comprising cases of both non-comunicating and communicating hydroceles. The study included observations on the nature, formation and reabsorption of hydrocele fluid, the surgical anatomy of the hydrocele sacs, the phenomenon of spontaneous cure, recurrence after orthodox surgical procedures and the results of operation by the Adelaide method. With regard to the nature of hydrocele fluid, Dr. McKay's finding was confirmed—that the fluid obtainable from either a communicating or a non-communicating hydrocele was indistinguishable from the peritoneal fluid which could be aspirated from a hernial sac. That fluid had a cell content of the order of 200 to 250 cells per cubic millimetre. The cells were predominantly lymphocytes with a few large mononuclear cells, probably epithelial in character, but no polymorphs. The protein content was of the order of three to four grammes *per centum* and the ratio of the different protein fractions was the same as that found in normal plasma. The mechanism of entry of that fluid into the hydrocele sacs was revealed in 50 cases by a careful operative study of the surgical anatomy. In those 50 cases, a communication between the hydrocele and the peritoneal cavity was demonstrated, although in many of those cases the communicating processus was so fine that it would have escaped a mere casual inspection of the cord for a hernial sac. In 11 cases, no macroscopic communication could be found. Broadly speaking, the cases could be grouped into four representative types, although there were all manner of variations and gradations between those types. First, there were those in which no hernial sac was present, but a fine patent *processus vaginalis* joined the hydrocele to the peritoneal cavity. In the second group, a small funnel-like hernial sac was present at the upper limit of that processus, and in the third group there was a rather extensive hernial sac extending for most of the length of the inguinal canal, or beyond, to communicate by a short, though very fine, processus with the hydrocele sac. In the fourth group an extensive hernial sac was present, which shared a common wall with the hydrocele sac, but no communication was demonstrable. It had to be emphasized however, that in all the cases studied a patency of at least some part of the *processus vaginalis* was present.

With regard to the mechanism of fluid formation in the 11 cases of non-communicating hydrocele, Dr. Fowler said that four possible ways could be considered. First, a previous patency of the *processus vaginalis* might have undergone delayed spontaneous obliteration. That was to him the most likely mechanism, as he thought the subsequent evidence would show. The second possibility was that patency of microscopic proportions might have been present and have escaped detection. However, that could not be the explanation of all those cases, because in two specimens serial sections were made of the excised common wall of the hernial and hydrocele sacs, and no communication was present. A third possibility was that peritoneal fluid from the hernial sac diffused into the hydrocele through the common wall, but the operation findings in one case of spontaneous cure, to be described, rendered that mechanism unlikely. The last possibility was that some local disturbance of the equilibrium between fluid production and fluid absorption across the hydrocele sac wall was the prime factor concerned. That, presumably, would be a situation akin to the adult idiopathic hydrocele of old men. That last possibility could be definitely excluded by the results of some experimental operative procedures which were undertaken. In four of the eleven cases of non-communicating hydrocele the fluid was deliberately left imprisoned in the hydrocele sac and a simple herniotomy was performed. In all four cases the hydrocele fluid subsequently reabsorbed. In the seven other cases of non-communicating hydrocele, a simple herniotomy was combined with emptying of the hydrocele fluid by needle aspiration or by a small stab incision. It was hard to believe that these small wounds of the hydrocele wall would not be rapidly repaired, thus allowing reaccumulation of fluid if local factors in the wall were responsible. As further evidence against the local formation theory there were two cases in which operative evidence was obtained of spontaneous obliteration of a patent *processus vaginalis* with subsequent disappearance of the hydrocele. In one of those cases, the sequence of events was such as to make the "percolation" theory unlikely, and it was therefore worth describing the history of that patient in some detail. He was a child in whom a hydrocele of the right spermatic cord had been observed on numerous visits to the out-patient department, and on one occasion five weeks prior to operation the lump measured two inches by one and a half inches. One week prior to operation he again attended the clinic, where it was noticed that the hydrocele had disappeared, but there was a palpable thickening of the cord. At operation, dissection of the cord revealed a very fine patent processus extending from the peritoneal cavity to about halfway down the inguinal canal, where it expanded somewhat into a small fusiform sac. Immediately below that little sac, and with no apparent communication with it, was a small blind sac containing virtually no fluid and measuring two centimetres by one and a half centimetres. That sac had thick vascular walls, which could not be readily dissected from the remaining cord structures; that suggested some recent inflammatory reaction. A distance of about half an inch separated it from a normal *tunica vaginalis*. Dr. Fowler said that the concept of the sequence of events in that case was that the fine patent processus initially supplying the hydrocele of the cord underwent spontaneous obliteration close to its junction with the hydrocele sac. What then followed was a reabsorption of fluid from the distal sac and a gradual distension of the then blind end of the patent processus above, to form the beginnings of a new hydrocele of the cord. If percolation of fluid from above was the means of perpetuating non-communicating hydroceles, then there was no reason why the hydrocele in that case should not have persisted. The evidence, therefore, was against all the theoretical mechanisms of the fluid formation except the first—that a communication with the peritoneal cavity was present at some stage in the life history of the hydrocele, and that that underwent spontaneous obliteration at one point along its course.

Summarizing the evidence up to that stage, Dr. Fowler said that in all the cases dealt with in the present series there was a patency of at least some part of the *processus vaginalis*; that fluid taken from both non-communicating and communicating hydroceles was indistinguishable from the peritoneal fluid obtainable from a hernial sac; that in those cases of non-communicating hydrocele in which simple herniotomy alone was performed, the hydrocele disappeared; and that in those cases of non-communicating hydrocele in which the fluid was emptied from the sac, simple herniotomy alone was sufficient to prevent its reaccumulation. It was his belief, therefore, that the evidence pointed strongly to the congenital origin of all non-secondary hydroceles in childhood, and that the origin of the hydrocele fluid was the peritoneal cavity.

Dr. Fowler went on to say that as far as treatment was concerned, if those postulates as to the congenital origin of hydroceles were accepted, then all that was required was to prevent the fluid from entering the hydrocele sac. Thus, a rational approach to treatment would be simply the ligation and severance of any patent section of the *processus vaginalis testis*, combined with needle aspiration of fluid from the hydrocele sac. Ignoring the sac itself in that fashion would avoid those complications which might follow the orthodox procedures. Troublesome scrotal œdema or hæmatomata were not infrequent complications of those operations, and in children there was the added risk of injury to the *vas deferens* or to the testicular vessels by the occasional operator. The procedures referred to were excision of a large sac or eversion of a smaller sac, the so-called Jaboulay procedure. Furthermore, those orthodox procedures were not immune from recurrence, and four cases of recurrence of that type were included in the present series. In those cases, careful dissection revealed a very fine patent *processus vaginalis* bringing the hydrocele into communication with the peritoneal cavity, and after the severance of that processus the hydroceles had not recurred. Various manœuvres might have to be resorted to in order to display that patent processus at operation. One was to apply manual pressure either to the abdomen or to the hydrocele sac. In either case it might be possible to force a few drops of fluid along the processus, in which case it would assume a characteristic distended, beaded, opalescent appearance. It could then be isolated as a separate structure entirely free from the other cord tissues. In the presence of a very large, tense hydrocele, the field of operation might be so crowded that the fine processus could easily be damaged by the dissection necessary to free the sac. Preliminary aspiration of the contents of the sac through the scrotal coverings would enable the dissection to proceed with greater facility. Another step which greatly facilitated the finding of the processus was the routine

division of the external oblique aponeurosis, giving an adequate access to the internal ring.

Finally, Dr. Fowler said that he wished to put before the meeting again the main conclusions which had emerged from the study. First, with the uncommon exceptions of secondary hydroceles, all hydroceles in infancy and childhood were congenital in origin. Secondly, all cases were associated with a patency of the *processus vaginalis testis* in at least some part of its extent. Thirdly, the ligation and division of that patent processus was all that was necessary to cure the condition. For the peace of mind of both surgeon and parents, it was a wise precaution to empty the sac by needle aspiration, which involved the minimum of trauma to the scrotum or its contents. Lastly, the treatment of recurrent hydrocele was to find the patent processus and to deal with it in the manner described.

Dr. R. Howard, in opening the discussion, said that he was interested in the neonatal hydroceles. He thought that there were two varieties. The first was the bilateral type, which usually cleared up spontaneously in three to eighteen months, and he presumed that in those the *processus vaginalis* became obliterated. The second variety was the tense, unilateral hydrocele, often painful. In two such cases he had been unable to find any communication, and the testis and epididymis were normal. He did not think that the hydrocele would have been tense if the fluid had come from the abdomen. Dr. Fowler said that he had seen only one child with a unilateral tense type of hydrocele causing pain. That was an abdomino-scrotal hydrocele, and operation was resisted. However, the hydrocele was becoming smaller. He thought the tenseness was due to the obliquity of entry of the processus into the sac. Tough fascial bands could form a valve and stop the fluid from going up until they were cut.

Dr. P. Jones asked what was the effect of a tense hydrocele on the after-development of the testis.

Dr. Fowler, in reply, said that he would not care to prognosticate. The testes looked normal on naked-eye examination, but in regard to long-term changes he could not speak.

Dr. D. Stephens said he thought that if a hydrocele was slack, it was in the process of resolution. If it was tense, it was a hydrocele with a valve. If a tense hydrocele became slack, then it would resolve, but not otherwise. They were all worth watching for several years.

Dr. Fowler said that he was not in complete agreement with Dr. Stephens that slackness was related to the imminence of spontaneous cure. He thought that in the first year there was no indication to operate, but thereafter most patients had large hernial sacs, and operation was carried out for that reason. However, pain would be an indication for operation.

Dr. M. Blanch, in closing the discussion, said that operation for hydrocele did not seem to be indicated unless there was pain or hernia, or unless spontaneous cure was delayed too long for the parents' peace of mind.

The Assessment of Ventilatory Capacity.

Dr. Bryan Gandevia discussed tests of ventilatory capacity and their clinical applications, first placing that group of tests in perspective in relation to the full range of tests of pulmonary function. He said that most tests of ventilatory capacity were in fact pieces of respiratory gymnastics rarely performed by the subject in the course of his normal activities, and it could be regarded almost as a fortunate coincidence that in general the results of those tests, especially if combined with estimates of the ventilatory response to exercise, correlated reasonably well with the extent of pulmonary disability. Indeed, one could go further and say that although functional tests did not offer specific pathological diagnoses, certain patterns of abnormality in ventilatory capacity tests—in other words, certain patterns of functional abnormality—could be related to various types of pathological processes, and quite accurately so if the tests were interpreted with a full knowledge of the clinical and radiological findings.

Dr. Gandevia went on to describe what he termed the single-breath tests of ventilatory capacity. He said that the simplest spirographic tests would be performed with a basal metabolic rate apparatus (Benedict-Roth); they included separate estimations of the vital capacity (made during a slow and unhurried expiration), the inspiratory capacity and the expiratory reserve volume. The presence of "air-trapping", such as occurred in emphysema and asthma, was suggested when the sum of the two last-mentioned exceeded the vital capacity. The presence of reversible bronchoconstriction was indicated by improvement in vital capacity after the administration of an aerosol of 1% isoprenaline for one to two minutes. With a fast-moving kymograph (paper speed of one to four centimetres per second) and an appropriately designed spirometer, more information was obtained by recording a tracing of the whole of a maximum forced expiration. From that "forced expiratory spirogram" the "forced vital capacity" and the "forced expiratory volume over the first second" were determined; normally the volume exhaled in the first second of a forced expiration exceeded 80% of the vital capacity, that figure being almost independent of age, build or sex. From the forced expiratory volume at one second it was also possible to calculate an indirect estimate of the maximum breathing capacity of that subject. The only advantage of the latter manœuvre was to present the findings in terms of a test with which most clinicians were familiar. Dr. Gandevia stressed the advantages of the forced single-breath tests over the maximum breathing capacity estimated in the conventional manner, the former being more repeatable, less tedious and less exhausting. The value for the maximum breathing capacity, as usually estimated, was also seriously influenced by the rate at which the patient happened to breathe. In general, in normal subjects and in patients with a restrictive type of ventilatory defect (which he would discuss later), the results for the maximum breathing capacity test tended to increase with increasing rate, whereas in patients with an obstructive type of ventilatory defect (for example, asthma), the values fell as the rate increased. The problems which those observations posed did not arise when the assessment of ventilatory capacity was based upon the analysis of the spirogram of a single breath. Two patterns of ventilatory defect emerged from the results of those tests: (i) an obstructive type of ventilatory defect, as in asthma and emphysema, characterized by a normal or low vital capacity, of which less than 75% (often only 30%) was expired in the first second, and by a low breathing capacity; (ii) a restrictive type of ventilatory defect, seen in ankylosing spondylitis (probably also in funnel chest), paresis of the respiratory muscles, diffuse pulmonary fibrosis and space-occupying lesions in the chest, and characterized by a lowered vital capacity, a proportionate reduction in the one-second volume so that the percentage expired in this time was normal, and a normal or nearly normal maximum breathing capacity (provided the latter test was carried out at a rate above about 60 breaths per minute). Occasionally evidence of both types of defect was found in the spirographic results.

Dr. Gandevia then discussed work done in collaboration with K. M. Hume on the change in ventilatory capacity following the administration of a bronchodilator aerosol. In asthmatic patients in severe *status asthmaticus*, the response was at first small, as reflected in the increase in vital capacity or forced expiratory volume at one second. The response increased steadily as the patient's wheeze diminished and his pre-aerosol ventilatory capacity improved until a certain maximum response was obtained. Thereafter, with further improvements in the patient's pre-aerosol ventilatory capacity, the response progressively diminished in such a way that the values for post-aerosol ventilatory capacity remained constant. In emphysema that pattern of response was less well defined. The curve of response against initial value appeared to be characteristic of and repeatable for any one patient. The implications of those observations were discussed, particularly in regard to maintenance therapy with cortisone and to the determinations of relative amounts of structural and functional abnormality.

Dr. Gandevia went on to point out that the significance of a given value for the maximum breathing capacity (whether obtained by direct or indirect method) was best assessed in the light of the patient's ventilatory requirement for a standard amount of exercise. Of two patients with a maximum breathing capacity of 80 litres per minute, one might perform a standard exercise test with a minute ventilation of 80 litres, while the other might require 50 litres. The first would probably not be unduly dyspnœic, but the second would feel extremely short of breath at the end of the test. An increased ventilatory cost of exercise was seen notably in association with impaired diffusing capacity for oxygen and in patients with stiff lungs as in pulmonary congestion.

Dr. Gandevia finally reviewed briefly some of the clinical applications of the tests. He said that while they had some part to play in diagnosis—for example, in the detection of clinically latent "bronchospasm"—their main significance lay in their provision of an accurate and objective estimate of the "bellows function" of the lungs, an estimate which, with certain reservations, correlated well with clinical disability. From the clinician's point of view, they had obvious applica-

tion in the assessment of progress and in the selection and evaluation of therapy in a wide variety of respiratory and other disorders.

DR. N. WETTENHALL asked how those tests would be applied in the Royal Children's Hospital, and what was the youngest age at which they could be used.

Dr. Gandevia, in reply, said that at the moment studies of normal children were being obtained. Children with funnel chests were being tested before and after operation. The tests might possibly be used as a research tool to follow the efficacy of certain lines of treatment—for instance, in asthmatics—or to gain more information on the fundamental physiology of various chest conditions. The tests might, for instance, be used to find whether some of the older patients with fibrocystic disease of the pancreas could be helped by bronchodilator therapy. Four years was probably the youngest age at which the test could be used.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE SYDNEY HOSPITAL.

[From the *Australasian Medical Gazette*, November, 1891.]

ONCE more an attempt has been made by exposing the shameful state of the Sydney Hospital to arouse the indignation of the public at the apathy which has marked the reception of all appeals hitherto made with the object of relieving this charity from the disadvantages under which it labours. The report of the Medical Superintendent of the Sydney Hospital has been published in all the daily papers of the city, and together with the comments thereon have thus been brought under the notices of every member of the community. The straits to which those who are treating the sick poor of the city are reduced by the inadequate and dangerous nature of the buildings in which these poor creatures lie at a time when it is above all desirable that they should receive every care that humanity can dictate are forcibly but temperately set forth in it. No excuse can be found which will absolve those who are in a position to deal with the scandalous and cruel facts therein detailed unless at the earliest possible moment steps be taken to remedy them. More than twelve months ago Parliament by a large majority expressed the determinations by resolution that the hospital buildings should be completed. The Public Works Committee nearly a year ago was engaged in enquiring into the circumstances connected with this question and finally made a report that this should be done. The condition of affairs at the Sydney Hospital reflects upon the character for humanity of every citizen of Sydney so long as this persistent delay and apathy are tolerated. The Medical Superintendent has deliberately stated that numbers of patients who should be admitted for treatment are refused from want of room. The medical staff corroborate this statement and intensify it by declaring that owing to his representations they are often obliged to discharge patients before they consider them fit to go. The officers of the institution in their reports have invited the most searching investigation into the accuracy of these assertions by anyone interested in the matter. Those gentlemen who in their evidence before the Public Works Committee or elsewhere have either ignored these statements or denied their truthfulness, apparently without personal knowledge of the facts, will bear the weight of heavy responsibility in the event of any disaster occurring as a result of further delay. We ourselves are convinced of the absolute necessity for the immediate completion of the building and for the provision of accommodation for the number of patients specified by the medical staff of the hospital in their report. At the same time we are deeply sensible of the discredit which attaches to us all from the permitted continuance of the existing state of things.

[Because its wards were considered to be inadequate and insanitary, parliamentary approval was granted for the demolition of the main building of Sydney Hospital in 1878, and the work was completed in the following year. The Governor, Lord Loftus, laid the foundation stone of the new building in July, 1881. Progress was, however, very slow. The Government of the day still hoped to utilize the site for new Law Courts, and it was further argued that the function of Sydney Hospital, apart from a casualty department, could be fully subserved by the new Prince Alfred Hospital. There was much acrimonious discussion, with several enquiries and numerous deputations, before necessary funds were made available, and the building was not formally opened by the Premier, Sir George Dibbs, until August, 1894.]

Correspondence.

ANÆSTHESIA IN HEART DISEASE.

SIR: Even if those addicted to excessive letter-writing might be suspected of paranoid tendencies, I feel bound in simple courtesy to acknowledge the comments made recently by Mr. M. P. Susman in the above connexion (M. J. AUSTRALIA, March 1, 1958). Undoubtedly reassurance, suggestion and even hypnosis have a place in such work, and indeed may sometimes be surprisingly helpful. As a rule, however, pharmaceutical aids are superior in this regard to psychological extravagances, especially when the latter prove to be inadequate.

It should not be forgotten that on September 30, 1846, one Eben Frost sought relief from toothache at the hands of W. T. G. Morton, dentist, of Boston, Massachusetts, U.S.A. An extraction being necessary, the hapless victim pleaded for mesmerism, but was given ether instead, with the most happy results for all mankind. Ever since then hypnotism has been of little real significance in the relief of surgical pain.

From recent correspondence in your Journal, sir, it would appear that if Mr. Susman could only succeed in hypnotising his anæsthetists all would indeed be well.

Yours, etc.,

S. V. MARSHALL.

"Harley",
143 Macquarie Street,
Sydney.
March 13, 1958.

EPISIOTOMY.

SIR: Might I offer my congratulations and thanks to Dr. Hoyle, of Inverell, for his carefully considered, well-balanced and ably expressed exposition of the subject of episiotomy? I feel aggrieved that I did not meet lecturers of his ability during my course at the University of Sydney. I am hoping that his letter will fall as manna from heaven into the hands of some groping students of sixth year. Such a happening will repay me somewhat for the ordeal of setting myself up as an Aunt Sally. Further, I would like to compliment the Collins Street correspondent upon the subtlety of his wit. I have appended his effusion to my scrapbook as an example of Gallic salt across the Murray. I must add, however, that this gentleman has not helped in our general search for knowledge. Nay, rather has he done much to dissuade doctors of more sensitive temperament from using THE MEDICAL JOURNAL OF AUSTRALIA as a means of testing their information and procedures at the bar of courteous constructive criticism. A quip, however brilliant, is surely unworthy of the ethics of medical correspondence.

Yours, etc.,

L. J. SHORTLAND.

"Otaki",
Marrickville,
New South Wales.
March 21, 1958.

SIR: Lest others more able have not opposed Dr. Shortland's and Dr. Hodgkinson's condemnation of episiotomy, I will. Firstly, the fragile head of a premature infant may be damaged against an unyielding primiparous perineum—all experienced in obstetrics must surely have been dismayed to lose such a baby, despite fœtal heart sounds being good throughout the second stage, through late intra-partum cerebral hæmorrhage revealed immediately after birth. Secondly, the management of a breech delivery, especially in a *primipara*, both for the breech and for the after-coming head, is facilitated by episiotomy. This can

be done, of course, and is best done with local anæsthesia or with a minimum amount of general anæsthesia (and I like chloroform). In emergency I have done episiotomies with no anæsthesia; done quickly with sharp scissors I believe it causes very little pain. Certainly less pain than the procedure taught us, as students, to digitally dilate the vaginal orifice. Surely Dr. Hodgkinson would have to give some general anæsthesia to dilate the introitus with obstetrical forceps, and a minimum of anæsthesia is the goal of good obstetrics.

Suturing of an episiotomy, because further tearing may make it an irregular wound, is slightly more tedious than closing the usual "tear". But every obstetrician must have been impressed with the lesser amount of vaginal injury which occurs with an episiotomy. Elimination of the wicked third degree tear is an obvious plea for judicious episiotomy. This is all standard teaching which hard experience has proven. There is no need to take the cobwebs off our textbooks for this cause. There is no place for puffed-out obstetrical pride at never getting a tear or having to do an episiotomy.

Yours, etc.,

158 Maitland Street, Narrabri, New South Wales. March 20, 1958.

HARTWELL WOOLFORD.

HYPERTENSION.

SIR: Your timely annotation about hypertension (March 1, 1958) will be welcomed by all who are familiar with the effects of over-treatment of this physical sign whose significance is doubtful. Symptomless hypertension as high as 220 is common in arthritic patients over 50 years. I have observed large numbers of them for many years, and the only casualty I can recall is an old lady who was carried off by hemiplegia after she had turned 80. Fry (*Brit. M. J.*, 1957, 2: 1453), reviewing five years in general practice, reports that 40% of his patients over 70 years have hypertension which is causing no symptoms. It is unwise to tell such a patient that she has high blood pressure. When they do complain of headaches, giddiness and other symptoms believed to be characteristic of hypertension, they obtain as much relief from placebos as from hypotensive drugs (Salassa and others). If the hypertension is accompanied by encephalopathy, no known drug or neurosurgical operation has been proved to influence the course of the disease. The patient is often extremely worried, and she will receive great benefit from a doctor who is sincerely interested in her, prescribes a non-toxic placebo and convinces her that everything possible is being done for her.

Yours, etc.,

410 Albert Street, East Melbourne, C.2. March 17, 1958.

MICHAEL KELLY.

BLOOD WANTED.

SIR: Your leader of March 8, 1958, underlines the importance of an adequate and continuing permanent source of blood wherever surgery, midwifery or medicine is practised. Without "blood" most of the recent advances in major surgery could not have taken place and many people living today must have died but for the blood transfusion services. It is only too obvious that the maintenance of an adequate supply of blood is essential to every hospital. What is not so obvious is the fact that every hospital can and should be self-sufficient as regards its blood needs.

Clearly a "couple o' bottles o' blood" afford much moral support to both surgeon and anæsthetist before and during a crisis, but the value of the "blood" to the patient does not compare with a pint of direct blood delivered when the need arises. Every hospital needs its own donor panel and its own Julian Smith pumps if it is to adequately care for hæmatemeses, post-partum hæmorrhages and severe burns. None of these cases should be treated with bottled blood.

Clearly, in some cases "bottled blood" is better than no blood at all, e.g., (i) in severe anæmia of gradual onset; (ii) in hæmorrhage, provided the bleeding point has been ligated. But in general blood by direct transfusion is many times better than citrated blood. Blood is not improved by being "dripped in". In fact blood is so valuable that no community can afford to citrate it, store it, process it or waste it. The belief that plasma is "good for burns" is sheer nonsense. Let us be honest about it. Is it not easier on the conscience to turn three pints of old blood into plasma than to tip them down the sink? In fact we cannot afford to do either.

If every hospital auxiliary regarded the organization of a donor panel as its most important duty, then every hospital would be independent in terms of blood. The dreadful stories of 24 pints of blood being poured down the drain via a bleeding ulcer would never be heard; burns cases would not be made to float in plasma which keeps oozing from them because of its citrate content; the post-partum hæmorrhage deaths would disappear; the never-ending appeal for blood would cease because the supply would have caught up with the demand.

For making this happy state of affairs possible this community is indebted to the late Dr. Julian Smith, whose blood transfusion pump makes citrated blood and the consequent appalling waste of blood no longer excusable.

Yours, etc.,

Mooroopna, Victoria. March 14, 1958.

D. G. MACKELLAR.

UNEXPLAINED SUDDEN DEATH.

SIR: At a coroner's inquest at Lithgow on the sudden death of a young woman the medical evidence was to the effect that the woman "just ceased to exist". Publication of details of this case would be welcomed. Do your readers know of any other instances when persons "just ceased to exist"?

Yours, etc.,

195 Macquarie Street, Sydney. March 17, 1958.

C. M. MCCARTHY.

Obituary.

LESLIE PROUD WAIT.

WE are indebted to Dr. Robert Southby for the following appreciation of the late Dr. Leslie Proud Wait.

The sudden passing of Leslie Proud Wait on October 28, 1957, caused a deep gloom throughout the Royal Children's Hospital, Carlton, to which institution he had rendered outstanding and faithful service as a member of the senior medical staff for a number of years. Les Wait was one of that group of specialists, nowadays rapidly diminishing, who had served a long apprenticeship in the unrivalled school of general practice preparatory to restricting his activities entirely to those of a physician. He was a man of outstanding character, and of the highest ideals to which he constantly adhered throughout his life. His first duty was always to the patients under his care, and to them he gave freely, and at all times, of his many talents, frequently carrying these cares with him far beyond the confines of the hospital. He was a sterling colleague, and one upon whom his fellow practitioners could always rely for a most considered and beneficial opinion in any abstruse clinical problem with which he was presented. He was never happier than when handling one of the little patients in his unit, with all of whom, and with the nursing staff, he was at all times a most sincere and valuable friend. With the parents of a seriously ill child his quiet and earnest deliberations were always a model of courtesy and tact and a tremendous strength to such parents in their time of trouble. He was in fact the personification of the truly Christian doctor.

Apart from his clinical work, Wait was an excellent organizer and administrator, and these qualities were seen to outstanding advantage during the period in which he served in the capacity of honorary secretary of the senior medical staff. His quiet and unassuming personality was ever the cloak of a strong and determined individual who would always fight to the last extreme for any principle which he regarded as right; in this respect the strength and determination of his character appeared at their best.

How happy is he born and taught
That serveth not another's will;
Whose armour is his honest thought,
And simple truth his utmost skill.

He was also an excellent teacher and an inspiration to his students who appreciated his efforts on their behalf, at the same time realizing that he had no time for inattention or inefficiency, and yet was most sympathetic to the conscientious and earnest among the embryo doctors whom he was delighted to instruct. His loss will be felt far beyond the boundaries of his loved hospital and his own field of practice, for he was a most popular member of the Australian Pædiatric Association, and his pædiatrician friends from other States will sadly miss his friendship and his wealth of experience, which were so evident at the meetings at Canberra.

Above all, Les Wait loved his wife and family and his home, and he was never happier than when in these surroundings with, in addition, his garden, and particularly his orchids, of which he was an enthusiastic and successful grower. The heartfelt sympathy of all his friends goes out to his widow and her two married daughters. We all feel that we have lost in person a most inspiring member of our team, but his spirit and his influence will live on in the institution which he served so devotedly for such a lengthy period. Of Les Wait it can be truly said:

> Lives of great men all remind us
> We can make our lives sublime
> And, departing, leave behind us
> Footprints on the sands of time.

A colleague who wishes to remain anonymous sends the following appreciation of Dr. Leslie Wait:

My friendship with Les Wait began over 40 years ago when we were schoolboys at Wesley College; later we were fellow medical students and subsequently he was my medical adviser. The friendship continued until his untimely death. Wait graduated M.B., B.S. (Melbourne) in 1925, and after a year as resident medical officer at Ipswich Hospital (Queensland) commenced general practice in North Balwyn, where he remained for some 20 years. While carrying on a busy general practice he obtained his M.D. in 1935, and then became associated with the Royal Children's Hospital, where he was appointed physician to out-patients in 1946 and physician to in-patients in 1951—an appointment he held until his death. Wait was an exemplar of all that is good in a sound general practitioner. He earned the respect and affection of his patients, and when he relinquished general practice to practise as a physician with a special interest in pædiatrics, the quality of his work exemplified the opinion of many, that the best consultant is the one who has come from the ranks of the family doctor.

WILFRED EVANS.

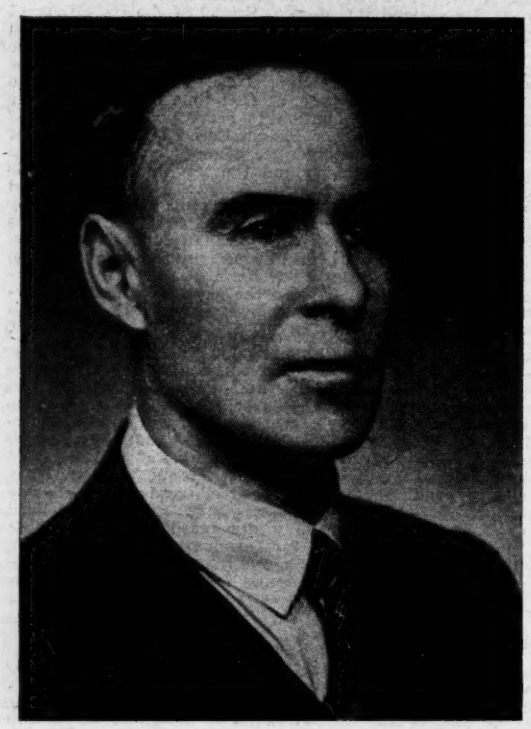
DR. WILFRED EVANS, who died on December 20, 1957, was born at Cooma on September 10, 1889. He had his early schooling locally, but later was enrolled at the Scots College, Sydney. He entered the Medical School of the University of Sydney, where he graduated in 1914, top of his year, with first class honours and the University medal. After the prescribed wartime short period of hospital residency he enlisted in the Australian Imperial Force and was posted to the 3rd Light Horse Brigade, at that time on Gallipoli. He continued after the evacuation to serve with the mounted troops until the end of the Middle East campaign and saw plenty of action. His ambulance commander regarded him as an "outstanding officer", and the war historian records that in the second Gaza engagement: "Captain Wilfred Evans worked all day under fire of guns and assisted by only four men handled no less than 240 wounded." He was later appointed D.A.D.M.S. at Australian Mounted Division Headquarters and took his full share in the anti-mosquito campaign against malaria in the Jordan Valley which Major-General Downes described as "the greatest effort in prevention of disease undertaken by the Medical Service in Palestine".

Evans's innate ability and particularly his versatility made him indeed a very valuable officer. In Allenby's final drive up the Maritime Plain the large number of sick and wounded prisoners presented quite a serious problem, and Evans was for some time "caretaker" C.O. of a German military hospital of 300 beds at Jenin. When the Australian troops entered Damascus they found 3000 enemy sick and wounded receiving most inadequate care, frequently in very unsanitary conditions, with little food, and practically no medical stores. Evans was appointed Principal Medical Officer of Damascus and rapidly introduced order into chaos; he was also responsible for evacuation of Australian casualties to the coast, which was promptly arranged. For his services in these campaigns Wilfred Evans was four times mentioned in dispatches and awarded a Military Cross.

When he was demobilized in 1920, Evans joined Dr. P. L. Hipsley in a busy practice at Waverley, which Dr. W. E. Kay entered when Dr. Hipsley retired in 1924. Dr. Archie Cunningham joined them in 1926. Evans had already in 1922 obtained an appointment as assistant physician at Sydney Hospital, where he had earlier been a student. For some years he held similar appointments at the Royal Alexandra Hospital for Children and at Lewisham Hospital, but relinquished them reluctantly as his responsibilities elsewhere increased. In 1928 he qualified for membership of the Royal College of Physicians of London and took up consulting practice. He was keenly interested in his out-patient work and particularly enjoyed teaching students who, in their turn, found his bright, vigorous approach to clinical medicine most stimulating. In 1937 he was appointed an honorary physician at Sydney Hospital, a post he retained until 1949. However, there was an intermission in this period when he was once more engaged in army service. When war broke out in 1939 he was anxious to serve overseas as a physician, and when his medical fitness for overseas service was doubted offered to serve in any other capacity. His offer was gladly accepted by Major-General Downes, under whom he had served in Palestine. He was appointed to the Medical Directorate at Army Headquarters and placed in charge of AMD2, the section of the Directorate responsible for hospitalization, medical standards, medical transport, medical boards and invaliding. Policy in regard to hospitals in Australia had been already largely determined, but an immense amount of detail work had still to be done, and many problems to be discussed in regard to base (now repatriation) and other hospitals that were to be built in each State. It was perhaps the most frustrating and irritating job in the Service, as the medical representative in these discussions, insisting all the time that clinical considerations be paramount, became the "buffer" for all types of complaints. Architects, engineers, builders and particularly treasury officials raised all sorts of difficulties and many of the problems were quite without precedent. These discussions went on for years, ranged all over the Commonwealth and extended to hospitals in New Guinea. However, Wilfred remained undaunted; with his keen insight he readily appreciated the varying problems. His wide medical

knowledge, sound common sense, tact, and ready cooperation won the respect and confidence of his collaborators and he did outstanding work under very difficult conditions, contriving to remain cheerful throughout. In 1945 he resumed his consultant practice. He was a Foundation Fellow of The Royal Australasian College of Physicians, and in 1946 was elected a Fellow of the Royal College of Physicians of London.

He took a very special interest in Sydney Hospital, where for some years he was senior physician. He was strong in support of any movement to improve teaching or other standards in the hospital and himself set a very high



example. He was most assiduous in his attendance at staff meetings, where his opinion carried great weight in defining hospital staff policy. Although he was removed from clinical work for some ten years of war service, he remained to the end a busy and successful practitioner, secure in the esteem and gratitude of his patients. His activities were not confined to his professional work—he was well and widely read, he played a good game of golf, he could discuss with equal zest the culture of his beatiful trees at Wentworth Falls, or more recently his sheep at Exeter. His advice was valued on the Board of his old school and in the committee of his club. All these interests were fitted into a well-organized life, but always subordinated to its main purpose—the practice of medicine.

Wilfred had a pleasant, kindly personality, a strong sense of humour, and with all his attainments was always unassuming. His was indeed a life of sterling purpose well and truly served.

Wilfred Evans married Dr. Heather Ross and was extremely happy in his home life. Their elder son is a resident medical officer at Sydney Hospital, the younger is in his final year of medicine. Their only daughter is an undergraduate in law.

DR. C. H. WESLEY writes: Wilfred Evans lived in his early youth on the south coast of New South Wales. His mother, a devout Presbyterian, moulded his character and ambitions. His father came of a line of famous Welsh medical men. Wilfred's step-father gave him much help and encouragement. He attended the Scots College, Sydney, as a boarder, and then entered the University of Sydney. During his course he developed acute rheumatism and lost a year, but graduated top of the year. He was on the staff of the Royal Prince Alfred Hospital until he joined the Australian Imperial Force, embarking on the troopship *Karoola* in June, 1915, with a number of his friends, Ernest Parry, Claude Tozer, Charles Parkinson and myself.

On arrival in Cairo he was attached to the First Australian General Hospital in Heliopolis. Here he attended some men of the *Karoola*, wounded at Lone Pine seven weeks from their enlistment; they fought well and bravely, though the only training received was aboard ship. Later, with a group including Sir Victor Horsley and Dr. John Storey, at Alexandria he embarked on the Indian Medical Services hospital ship *Seang Choon* and proceeded to Gallipoli. The officers sent us ashore with good wishes and plenty of chocolate. After landing at Anzac, he joined the Third Australian Light Horse Field Ambulance commanded by Lieutenant-Colonel Rupert Downes. He was later detailed to the Ninth Light Horse on Rhododendron Spur as regimental medical officer. At the evacuation of Gallipoli, the remnants of the Third Light Horse Brigade left Gallipoli in "beetles" and boarded the *Abbassia*, a Greek ship on which the fare consisted of four items: stout, coffee, bacon and eggs, bread and butter—wonderful luxury after Gallipoli. The Third Light Horse Brigade with troops from Suvla Bay overcrowded H.M.T. *Karoo* on embarkation at Lemnos. Amongst the British troops was Lieutenant-Colonel Evans, of the Royal Army Medical Corps, his cousin. The *Karoo* arrived in Alexandria before rumour of the evacuation and before preparation of a camp on the Heliopolis Racecourse for the Light Horse Division. After the battle of Romani, 1916, he returned to the Third Australian Light Horse Field Ambulance. He was held in great esteem by the commanding officers, officers and men of the Ninth. Later he was to show marked executive ability as a staff officer.

Recently one of his old friends said: "Wilfred Evans is as charming as when I first met him as a young man." His was the charm which comes from kindness, consideration for the opinions and feelings of others, and humility in spite of great achievement and ability. He never showed disdain or patronage to his less able fellows. His fine character outshone his brilliant intellect.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

GENERAL REVISION COURSE, MAY 5 TO 16, 1958.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the annual general revision course will be held in Sydney for two weeks, beginning May 5, 1958. As in the previous course, the main emphasis is on therapeutics, and in addition the programme, as in former years, is a comprehensive survey of modern trends in diagnosis and treatment of special value to general practitioners. Members of the course are invited to bring their own X rays for the X-ray conference to be held on Friday, May 9.

Monday, May 5, in the Stawell Hall, 145 Macquarie Street, Sydney: 9.15 a.m., registration; 9.45 a.m., opening of courses; 9.55 a.m., election of chairman; 10 a.m., review of course; 10.10 a.m., group photograph of members; 10.45 a.m., "Penicillin-Resistant Staphylococci", Dr. Edgar Thomson; 11.45 a.m., "Geriatrics in General Practice", Dr. G. S. Procopis; 2.15 p.m., symposium on "Aspects of Biliary Tract Disease", Professor C. R. B. Blackburn (chairman); 2.15 p.m., "Diagnostic Methods and Their Indications", Professor C. R. B. Blackburn; 2.35 p.m., "Cholecystitis", Dr. Alan McGuinness; 2.55 p.m., "Obstructive Jaundice and its Diagnosis", Dr. D. W. Piper; 3.45 p.m., "Stone in the Common Duct", Dr. Stanley Goulston; 4.05 p.m., "Post-Cholecystectomy Syndrome", Dr. A. W. Morrow; 4.25 p.m., discussion.

Tuesday, May 6, in the Stawell Hall, 145 Macquarie Street, Sydney: Symposium, "Common Skin Diseases in General Practice", chairman, Dr. Ewan Murray-Will: 9.15 a.m., "The Management of Dermatitis of the Hands", Dr. F. J. Collett; 9.40 a.m., "Tinea of the Scalp", Dr. C. P. Reilly; 9.55 a.m., "Some Points About Pruritus", Dr. Adrian Johnson; 10.45 a.m., "The Treatment of Infantile Eczema", Dr. J. M. Rae; 11.05 a.m., "New Drugs in Dermatology", Dr. R. B. Perkins; 11.25 a.m., discussion. 11.45 a.m., "Gout", Dr. Selwyn Nelson; 2.15 p.m., "Is it Angina?", Dr. Alan McGuinness; 3.45 p.m., "What the General Practitioner Should Know About Automation", Dr. A. Denning.

Wednesday, May 7, in the Stawell Hall, 145 Macquarie Street, Sydney: 9.15 a.m., "Obesity in Male and Female", Dr. T. M. Greenaway; 10.45 a.m., "Tetanus", Dr. Keith Harrison; 11.40 a.m., "The Diagnosis and Treatment of Cerebral Vascular Diseases", Dr. George Selby; 12.20 p.m., "Control of Epilepsy", Dr. Eric Susman. Free afternoon: This afternoon has been kept aside for private arrangements and other specially arranged activities. A choice is offered to members to visit the following: mechanical brain "Silliac"; modern automation plant; Channel 9 Television Station; isotope laboratory, St. Vincent's Hospital; artificial kidney, Sydney Hospital; Recovery and Resuscitation Unit, Sydney Hospital; equipment for by-pass cardiac surgery (heart-lung machine), Royal Prince Alfred Hospital; Cobalt Beam Therapy Unit, Royal Prince Alfred Hospital; Unit of Clinical Investigation, The Royal North Shore Hospital of Sydney. 8.15 p.m., symposium, "New Drugs and Therapeutic Measures", chairman, Dr. C. G. McDonald: "Anticoagulant Therapy", Dr. Richmond Jeremy; "Tranquillizing Drugs", Professor W. H. Trethowan; "Recent Work in ACTH and Cortisone", Dr. A. W. Morrow; "Antidiabetic Drugs", Dr. Hales Wilson; "Hypotensive Therapy", Dr. James Isbister.

Thursday, May 8, in the Stawell Hall, 145 Macquarie Street, Sydney: Symposium, "Psychiatric Problems in General Practice", chairman, Dr. W. S. Dawson: 9.15 a.m., "Recognition and Treatment of the Depressive State", Dr. Cedric Swanton; 9.55 a.m., "Sexual Problems in Marriage", Dr. B. Peterson; 11.05 a.m., "Causes and Treatment of Insomnia", Dr. Ian Simpson; 11.40 a.m., "Acute Confusional States", Dr. D. C. Maddison; 12.15 p.m., discussion. In the Broughton Hall Psychiatric Clinic, Leichhardt: 2.15 p.m., demonstration of psychiatric problems in general practice by Professor W. H. Trethowan and the staff.

Friday, May 9, in the Stawell Hall, 145 Macquarie Street, Sydney: 9.15 a.m., "Management of Coronary Insufficiency", Dr. Frank L. Ritchie; 10.45 a.m., X-ray conference, Dr. Selwyn Nelson (chairman); speakers, Dr. Marjorie Dalgarno, Dr. P. H. Greenwell; 11.45 a.m., film session.

Saturday, May 10, in the Red Cross Blood Transfusion Centre, 1 York Street, Sydney: 9.30 a.m., demonstration of blood grouping, Rh testing and blood transfusion techniques, Dr. Hugh K. Ward and Dr. R. J. Walsh.

Monday, May 12, in the main lecture theatre, Royal Alexandra Hospital for Children, Camperdown: 9.15 a.m., "Everyday Emergencies in Domiciliary Pædiatrics", Dr. J. M. Alexander; 10.45 a.m., "Pædiatric Neurosurgery in General Practice", Dr. M. Sofer Schreiber; 11.45 a.m., "Demonstration of Various Practical Problems", Dr. John Harley; 1 p.m., lunch-hour discussions, held in the nurses' tearoom, with lecturers and senior members of the honorary medical staff, under the supervision of Professor Lorimer Dods; 2.15 p.m., "Recognition of Surgical Conditions in the Newborn", Dr. A. C. Bowring; 3.30 p.m., "The Management of Asphyxia in the Newborn", Dr. S. E. J. Robertson.

Tuesday, May 13, in the Stawell Hall, 145 Macquarie Street, Sydney: Symposium, "Gynæcological Problems in General Practice", chairman, Dr. George Stening: 9.15 a.m., "Management of Female Climacteric", Dr. J. Greenwell; 10 a.m., "Early Diagnosis of Carcinoma of the Cervix", Dr. Malcolm Coppleson; 11.15 a.m., "Aspects of Endometriosis", Dr. R. C. Gill; 12 noon, questions and answers; symposium, "The General Practitioner and His Obstetric Problems", chairman, Professor Bruce Mayes: 2.15 p.m., "Uterine Dysfunction", Dr. W. D. Cunningham; 2.40 p.m., "Management of Complications of Labour Causing Fœtal Distress", Dr. R. B. C. Stevenson; 3 p.m., "Radiation Injury to Fœtus or Mother during Pregnancy", Dr. E. A. Booth and Dr. D. R. Sheumack; 3.50 p.m., "Hypofibrinogenæmia in Pregnancy", Dr. J. M. Farrar; 4.10 p.m., "Any Questions?".

Wednesday, May 14, in the Stawell Hall, 145 Macquarie Street, Sydney: 9.15 a.m., "Ophthalmology in General Practice", Dr. R. Hertzberg; 10.45 a.m., symposium, "Anæsthetic Emergencies", chairman, Dr. L. T. Shea: "Respiratory", Dr. Douglas Joseph; "Cardio-Vascular", Dr. W. Shaw; "Miscellaneous", Dr. R. B. Speirs; discussion; 2.15 p.m., "Otitis Externa and Otitis Media", Dr. B. B. Blomfield; 3.45 p.m., "Pathology for the General Practitioner", Dr. C. B. Cox.

Thursday, May 15, in the Stawell Hall, 145 Macquarie Street, Sydney: 9.15 a.m., "Infant Feeding", Dr. Clair Isbister; 10.45 a.m., "The Ischæmic Limb", Dr. Alan Sharp; 11.45 a.m., "Fractures and Other Orthopædic Disabilities", Dr. Denis Rowe; 2.15 p.m. to 4.45 p.m., symposium, "The Care and Cure of Herniæ", chairman, Professor John Loewenthal: "Inguinal Herniæ", Dr. T. F. Rose; "Femoral Herniæ", Dr. C. E. Winston; "Herniæ in Childhood", Dr. J. Steigrad; "Trusses, Corsets and Appliances", a surgical appliance maker; "Legal Aspects", Mr. Antony Larkins, Q.C.; "Umbilical and Incisional Herniæ", Dr. N. R. Wyndham.

Friday, May 16, in the Stawell Hall, 145 Macquarie Street, Sydney: 9.15 a.m. to 11.45 a.m., panel discussion on "Problems in Medical Practice": moderator, Dr. V. M. Coppleson (honorary director); special panel, Dr. George Cummins, Dr. K. A. McGarrity, Dr. Ralph Reader; course panel, six members of the course will be chosen to act as monitors.

Social Activities.

Social activities during the course will include a cocktail party at the Hotel Australia on Tuesday, May 6, at 5.30 p.m., and a cellar party on Tuesday, May 13, at 5.30 p.m.

The Post-Graduate Golf Cup competition will be played at the Royal Sydney Golf Club, Rose Bay, Sydney, on Friday, May 9, from 12.30 p.m. The Brydon Cup will be awarded to the country member attending the general revision course who obtains the best score.

Post-Graduate Oration, 1958.

In the presence of His Excellency the Governor of New South Wales, the eleventh annual post-graduate oration will be given by Dr. Marjory Little on "Some Pioneer Medical Women of Sydney University" on Wednesday, May 14, 1958, at 8.15 p.m. in the Great Hall of the University of Sydney.

Fees and Method of Enrolment.

Fees for attendance are: full course, £12 12s.; mornings or afternoons only, £6 6s.; one week only, £6 6s. Early application, enclosing remittance, should be made to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney."

Fees and travelling expenses for this course are taxation deductions. When such deductions are claimed, "Taxation—File No. AF/1865" should be quoted.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

Special Course in Cardio-Pulmonary Physiology.

The Committee will conduct the following series of lecture discussions on the scientific basis of medicine, designed to present a survey of modern concepts in the field of cardio-pulmonary physiology in the light of recent research work: May 2, "Vascular Responsiveness in Hypertension", Dr. A. Doyle; May 23, "Œdema and the Problem of Aldosterone Production", Dr. D. A. Denton; July 4, "The Control of Body Fluid Volume", Dr. T. E. Lowe; August 1, "Pulmonary Function Studies in Clinical Investigation", Dr. B. H. Gandevia; September 5, "Some Aspects of Myocardial Ischæmia", Dr. A. G. Goble; October 3, "Physiological Aspects of Ventricular Septal Defects", Dr. J. M. Gardiner.

These lectures will be given at the Royal Melbourne Hospital, and all members of the medical profession are invited to attend without fee. Supper will be served at the close of the discussions.

Royal Children's Hospital Post-Graduate Pædiatric Week.

The Royal Children's Hospital Post-Graduate Pædiatric Week will be held from September 1 to 5. The programme will include lectures, presentation of short papers, demonstrations and ward rounds. The B.M.A. clinical meeting will also be held at this hospital on the evening of Wednesday, September 3.

PRINCE HENRY'S HOSPITAL, MELBOURNE.

Post-Graduate Course in Medicine, 1958.

THE honorary medical staff of Prince Henry's Hospital, Melbourne, will conduct a post-graduate course in medicine suitable for candidates studying for the degree of M.D. (Melbourne) and M.R.A.C.P. This will run for eight weeks from May 26 to July 19, 1958. Each day, sessions will be held from noon to 1 p.m. and from 2 p.m. to 5.15 p.m. Sessions will also be held from 9.30 a.m. to noon on Saturdays. It will be the privilege of the hospital to have Dr. M. D. Milne, M.D., M.R.C.P., physician and lecturer at the Post-Graduate Medical School, London, as a visiting

lecturer in the last four weeks of the course. Four afternoons a week will be devoted to lectures and case presentations. Members of the class will present to the physician cases examined in the mornings. Discussion of the case will follow and the physician will outline salient features demonstrated. Sometimes this procedure will be varied by the physician in charge by clinico-pathological presentations of cases from his ward or out-patient clinic. Didactic lectures followed by discussion will occupy the latter part of these afternoons. Wednesday afternoons will be devoted to pathology specimens presented by the pathologist and demonstrations in certain branches of medicine. The morning sessions will be occupied by pathology, ophthalmology, dermatology, electrolyte physiology, allergy, industrial medicine, medical radiology, electro-cardiography etc. For Saturday mornings, eight seminars on medical subjects have been arranged. Members of the panel taking part will each present short papers, to be followed by general discussion. Post-graduates will be advised of other teaching activities within the hospital—general and specialist out-patient clinics etc.

Enrolments, together with the fee of £15 15s., should be sent to the Melbourne Medical Post-Graduate Committee, 394 Albert Street, East Melbourne, by May 12, 1958.

Royal Australasian College of Surgeons.

FINAL FELLOWSHIP EXAMINATION.

A MEETING of the Court of Examiners for the final examination for Fellowship of the Royal Australasian College of Surgeons will be held in Perth, beginning on Saturday, August 16, 1958. Candidates who desire to present themselves at this examination should apply, on the prescribed form, to the Censor-in-Chief for permission to do so before July 3, 1958. The appropriate forms are available from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1. Candidates who have already been approved by the Censor-in-Chief, in general surgery and orthopædics, but who have not yet presented for the examination, may present for this examination provided they notify the Secretary of their intention to do so by July 3, 1958. It is stressed that entries close on this date and that late entries cannot be accepted. The examination fee is £26 5s., *plus* exchange on cheques drawn on banks outside Melbourne, and must be paid to the Secretary by July 3, 1958.

The examination will be conducted in general surgery and in the special branch of orthopædic surgery.

At its meeting held on June 23 and 24, 1956, the Council decided that until December 31, 1958, Fellows of other Colleges with which the Royal Australasian College of Surgeons has reciprocity of primary examinations and who obtained their Fellowship prior to January 1, 1950, may, at the discretion of the Council, be permitted to undergo a modified type of final examination. The conditions set out above regarding method of application for permission to present, date on which entries close, examination fee, etc., also apply to the temporary modified type of final examination.

PRIMARY EXAMINATION FOR THE F.R.A.C.S.

A PRIMARY EXAMINATION in anatomy (including normal histology) and applied physiology and the principles of pathology will be conducted in Melbourne, Sydney and Dunedin in September, 1958.

Written papers will be held simultaneously in the three cities on Thursday and Friday, September 4 and 5, 1958. The examiners will visit the three centres for the purpose of conducting the viva-voce section of the examination.

The examination is reciprocal with the primary examinations for Fellowship of the Royal College of Surgeons of England, the Royal College of Surgeons of Edinburgh, the Royal College of Surgeons in Ireland, and the Royal Faculty of Physicians and Surgeons of Glasgow.

Each examination is open to graduates of not less than one year's standing of a medical school approved by the Council of the College for the purpose.

Candidates must submit evidence of their qualification and of the date of acquirement thereof.

Forms of application for admission to the examination may be obtained from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

When entering for the examination, candidates must state whether they desire to appear before the Board of Examiners in Melbourne, Sydney or Dunedin.

The fee for admission or readmission to the examination is, in the case of the examination in Melbourne and Sydney, £26 5s. (*plus* exchange on cheques drawn on banks outside Melbourne). The fee for the examination in New Zealand is £26 5s. (New Zealand currency), and should be remitted by bank draft drawn on Melbourne in favour of the Royal Australasian College of Surgeons. The fee must be forwarded with the form of application so as to reach the Secretary at his office in Melbourne not later than July 24, 1958. It is stressed that entries close at the College office in Melbourne on July 24, 1958, and that late entries cannot be accepted.

FACULTY OF ANÆSTHETISTS: PRIMARY EXAMINATION.

A PRIMARY EXAMINATION for the F.F.A., R.A.C.S., in anatomy, physiology, pharmacology and pathology will be conducted in Melbourne, Sydney and Dunedin in September, 1958.

Written papers will be held simultaneously in the three cities on Thursday and Friday, September 4 and 5, 1958. The examiners will visit the three centres for the purpose of conducting the viva-voce section of the examination.

The examination is open to graduates of not less than one year's standing of an approved medical school.

Candidates must submit evidence of their qualification and of the date of acquirement thereof.

Forms of application for admission to the examination may be obtained from the Secretary, Faculty of Anæsthetists, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

When entering for the examination, candidates must state whether they desire to appear before the Board of Examiners in Melbourne, Sydney or in Dunedin.

The fee for admission or readmission to the examination is, in the case of the examination held in Melbourne and Sydney, £26 5s. (*plus* exchange on cheques drawn on banks outside Melbourne). The fee for the examination held in New Zealand is £26 5s. (New Zealand currency), and should be forwarded by bank draft drawn on Melbourne in favour of the Royal Australasian College of Surgeons Trust Account. The fee must be forwarded with the form of application so as to reach the Secretary at his office not later than July 24, 1958.

It is stressed that entries close at the Faculty office in Melbourne on July 24, 1958, and that late entries cannot be accepted.

College of General Practitioners.

THE Australian College of General Practitioners was incorporated under the provisions of the *New South Wales Companies' Act* on February 4, 1958, and the first meeting of the Interim Council of the College was held in Hobart on March 7, 1958.

The officers and members of the Interim Council are: Dr. W. A. Conolly (New South Wales), Chairman; Dr. David Zacharin (Victoria), Deputy Chairman; Dr. H. M. Saxby (New South Wales), Honorary Secretary and Acting Honorary Treasurer; Dr. B. N. Adsett (Queensland); Dr. Colin Anderson (Western Australia); Dr. R. D. Bartram (Victoria); Dr. D. M. Clement (Western Australia); Dr. A. C. D. Corney (Tasmania); Dr. H. E. H. Ferguson (Western Australia); Dr. W. J. Hamilton (Queensland); Dr. T. C. James (Tasmania); Dr. C. C. Jungfer (South Australia); Dr. M. O. Kent Hughes (Victoria); Dr. D. K. Kumnick (South Australia); Dr. G. D. McDonald (Victoria); Dr. L. R. Mallen (South Australia); Dr. H. S. Patterson (Queensland); Dr. J. G. Radford (New South Wales); the Honourable R. J. D. Turnbull (Tasmania); Dr. Colin Warburton (New South Wales).

A cablegram received from Dr. John Hunt, Honorary Secretary of the College of General Practitioners in London, read as follows: "Best wishes from Council to Australian College of General Practitioners. Suggest changeover July first for members British College." Officers and members of existing Faculty Boards have been invited to accept membership of the Australian College, and as from July 1, 1958, they will become the Faculty Boards of the newly established College. Invitations are to be issued to members of the College of General Practitioners to join the Australian College, and it is anticipated that the first general meeting will be held in Melbourne in September, 1958.

Congresses.

WORLD CONGRESS ON FERTILITY AND STERILITY.

THE third World Congress on Fertility and Sterility, sponsored by the International Fertility Association, will be held in Amsterdam, Holland, from June 7 to 13, 1959. The general outlines of the sections of the programme will be as follows: (i) Female sterility (physiology of reproduction, pathology, endocrinology, clinical problems, treatment). (ii) Male sterility (physiology of reproduction, pathology, endocrinology, clinical problems, treatment). (iii) Basic research and/or animal reproduction. (iv) Psycho-sexual problems.

Although any original report on some phase of fertility and infertility, either clinical or in the field of the basic sciences, will be considered, definite priority will be given to those papers concerning the following subjects: (i) Embryonic death (ætiology, pathogenesis, placental structures in its relation to the condition of the fœtus, functional problems and diagnosis of the embryonic and fœtal death, and habitual abortion). (ii) Hormonal factors and vitamins in fertility and sterility (ovulation and sterility, induction of ovulation, influence of thyroid, steroids etc. on ovulation, influence and physic-pathological significance of vitamins, hormones and spermatogenesis, vitamins and spermatogenesis). (iii) Relative value of the techniques for study of the endocrine functions in human sterility (study of the œstrogen function, study of the luteal function, study of the adeno-hypophysis function, testicular biopsy value, study *in vitro* of the fertilization of mammalian and human ova, use of radio-isotope designers in the study of the sexual function). (iv) Biochemistry of spermatogenesis. (v) Psycho-sexual problems in sterility.

For further information and registration, apply to Dr. L. I. Swaab, Honorary Secretary, Third World Congress on Fertility and Sterility, Sint Agnietenstraat 4, Amsterdam-C.

Honours.

NEW YEAR HONOURS.

IN the announcement of the names of Australian medical practitioners included by Her Majesty the Queen in the New Year Honours list we regret that the name of Professor Lambert Charles Rogers was overlooked. Professor Rogers was created a Commander of the Most Excellent Order of the British Empire.

Medical Appointments.

Dr. Kathleen A. Maros has been appointed Research Assistant in the Institute of Medical and Veterinary Science, Adelaide.

Dr. H. P. Duke has been appointed medical officer in the Mental Hospital, Ipswich, Queensland.

Dr. D. F. Potter has been appointed medical officer in the Brisbane Mental Hospital, Goodna, Queensland.

The following have been appointed members of the Council of the Queensland Institute of Medical Research: Dr. A. Fryberg, Dr. J. I. Tonge, Dr. A. D. D. Pye, Dr. G. C. Taylor and Dr. D. A. Henderson.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MARCH 15, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	1	4	..	1(1)	..	..	..	6
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	13(13)	..	..	1(1)	..	2	..	16
Diphtheria	1(1)	1(1)	..	..	..	..	..	..	2
Dysentery (Bacillary)	..	2(2)	..	..	1(1)	..	3	..	6
Encephalitis	1(1)	1(1)	..	..	..	..	..	..	2
Filariasis	..	..	1	..	..	..	..	..	1
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	34(16)	23(7)	20	2(2)	30(2)	..	2	..	120
Lead Poisoning	..	..	1(1)	..	..	..	..	..	1
Leprosy	..	..	..	..	1	..	1	..	2
Leptospirosis	1	..	1	..	..	..	..	..	2
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	2(2)	..	1	..	..	..	..	..	3
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1(1)	..	..	..	..	..	..	..	1
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	14(10)	..	2(2)	16(12)	..	..	..	32
Salmonella Infection	..	..	..	1(1)	3(3)	..	..	..	4
Scarlet Fever	16(9)	10(9)	1(1)	1	4(4)	..	..	..	32
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	2	..	..	..	3
Trachoma	..	..	..	..	58	..	..	..	58
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	23(8)	9(6)	10(7)	4(3)	10(8)	4(3)	2	..	62
Typhoid Fever	2(2)	1(1)	..	..	2(1)	..	..	..	5
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	1(1)	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Sheilshear, Michael Francis, M.B., B.S., 1955 (Univ. Sydney), 19 Mackenzie Street, Lindfield, New South Wales.

Monaghan, William, M.B., B.S., 1956 (Univ. Sydney), 25 William Road, Riverwood, New South Wales.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Chapman, Robert John (provisionally registered), M.B., B.S., 1958 (Univ. Sydney); Clumeck, Jean Marie (provisionally registered), M.B., B.S., 1958 (Univ. Sydney); Craigie, David John (provisionally registered), M.B., B.S., 1958 (Univ. Sydney); Russell, Diana Murchison (provisionally registered), M.B., B.S., 1958 (Univ. Sydney); Wong, Siew Poh (provisionally registered), M.B., B.S., 1958 (Univ. Sydney); Castaldi, Peter Anthony, M.B., B.S., 1957 (Univ. Sydney); Cooper, Ian Allan, M.B., B.S., 1957 (Univ. Sydney); Fraser, Donald Ivor Alexander, M.B., B.S., 1957 (Univ. Sydney); Taylor, Roy Francis le Cappelaine, M.B., B.S., 1957 (Univ. Sydney); Way, Maxwell Charles, M.B., B.S., 1957 (Univ. Sydney); Dagger, Victor Ross, M.B., B.S., 1956 (Univ. Sydney); Miles, Peter James, M.B., B.S., 1953 (Univ. Sydney); Parnell, Peter Montague, M.B., B.S., 1956 (Univ. Sydney); Sheldon, Bruce Henry Gilbert, M.B., B.S., 1955 (Univ. Sydney); Speechley, Ronald Alwyn, M.B., B.S., 1956 (Univ. Sydney); Ackermann, Isidor Anthony, M.D., 1940 (Univ. Bucharest), registered under Section 17 (1c) of the *Medical Practitioners Act*, 1938-1957; Goldner-Reiss, Irma, M.D., 1938 (Univ. Vienna), registered under Section 17 (1c) of the *Medical Practitioners Act*, 1938-1957; Levitski, Aleksander, M.D., 1923 (Univ. Tartu), registered under Section 17 (2A) of the *Medical Practitioners Act*, 1938-1957; Saave, Jan Jerzy, M.D., 1948 (Univ. Marburg), registered under Section 17 (2B) of the *Medical Practitioners Act*, 1938-1957; Simkovics, Gusztav, M.D., 1939 (Univ. Debrecen), registered under Section 17 (1c) of the *Medical Practitioners Act*, 1938-1957; Wilcox, Eva, M.D., 1949 (Univ. Bonn), licensed under Section 21C of the *Medical Practitioners Act*, 1938-1957.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Sharma, Romesh, M.B., B.S., 1954 (Univ. Adelaide), 88 Hutt Street, Adelaide.

Heaysman, Clive, M.B., B.S., 1957 (Univ. Adelaide), 250 Seaview Road, Henley Beach, South Australia.

Taylor, Benjamin John, M.B., B.S., 1945 (Univ. Adelaide), Kapunda Road, Freeling, South Australia.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Gunning, Julienne, M.B., B.S., 1957 (Univ. Adelaide); Pickering, T. G., M.B., B.S., 1957 (Univ. Adelaide); Parks, V. J., M.B., B.S., 1957 (Univ. Adelaide); Hobbs, W. H., M.B., B.S., 1957 (Univ. Adelaide); Allen, J. B., M.B., B.S., 1957 (Univ. Adelaide); Mann, W. E., M.B., B.S., 1957 (Univ. Adelaide); Muecke, D. S., M.B., B.S., 1957 (Univ. Adelaide); May, J. S., M.B., B.S., 1957 (Univ. Adelaide); Beare, J. H., M.B., B.S., 1957 (Univ. Adelaide); Gale, A. E., M.B., B.S., 1957 (Univ. Adelaide); Hollis, Yvonne, M.B., B.S., 1956 (Univ. Adelaide); Mocatta, Frances A., M.B., B.S., 1943 (Univ. Sydney).

Deaths.

The following deaths have been announced:

Watt.—Janet Paterson Watt, on March 10, 1958, at Penshurst, Victoria.

Wilson.—Thomas George Wilson, on March 15, 1958, at Adelaide.

Oxenham.—Humphrey Bede Oxenham, on March 17, 1958, at Wellington, New Zealand.

Bean.—Alan Reid Bean, on March 21, 1958, at Perth.

Sheilshear.—Joseph Lexden Sheilshear, on March 22, 1958, at Sydney.

Diary for the Month.

April 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
April 10.—New South Wales Branch, B.M.A.: Public Relations Committee.
April 10.—Queensland Branch, B.M.A.: Council Meeting.
April 11.—Queensland Branch, B.M.A.: General Meeting.
April 11.—Tasmanian Branch, B.M.A.: Branch Council.
April 12.—Queensland Branch, B.M.A.: Convocation.
April 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
April 16.—Western Australian Branch, B.M.A.: General Meeting.
April 17.—New South Wales Branch, B.M.A.: Clinical Meeting.
April 17.—Victorian Branch, B.M.A.: Executive Meeting.
April 18.—New South Wales Branch, B.M.A.: Ethics Committee.
April 22.—New South Wales Branch, B.M.A.: Hospitals Committee.
April 23.—Victorian Branch, B.M.A.: Council Meeting.
April 24.—New South Wales Branch, B.M.A.: Branch Meeting.
April 24.—South Australian Branch, B.M.A.: Listerian Oration.
April 24.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

All articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary is stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 *per annum* within America and foreign countries, payable in advance.

ILLUSTRATIONS TO THE ARTICLE BY H. J. RICHARDS AND K. VINER SMITH.

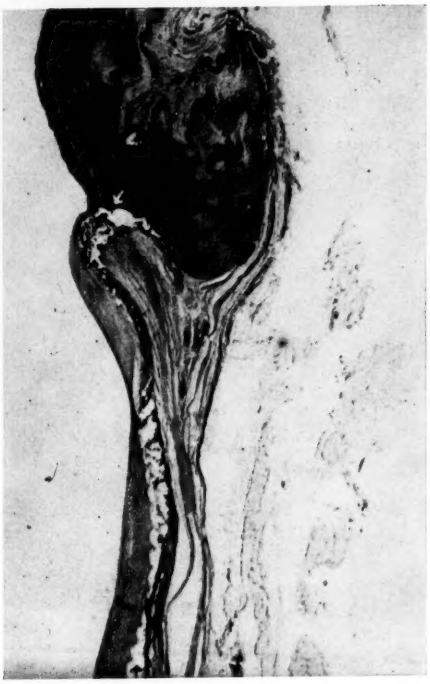


FIGURE III.

The junction of the prosthesis and the aorta after 86 days. The prosthesis has been dissolved out before the section was cut. Its position is indicated by arrows. Fibrous tissue is seen on each side of it, being especially thick in the angle between the prosthesis and the cut end of the aorta. (Hæmatoxylin and eosin stain, × 13.)

FIGURE IV.

This illustrates in higher magnification the tissue on the lower edge of Figure III inside the prosthesis. The fibrous tissue advancing from the top has passed under some fibrin. The endothelium has continued on over it for a short distance. (× 300.)

ILLUSTRATIONS TO THE ARTICLE BY P. A. TOMLINSON.

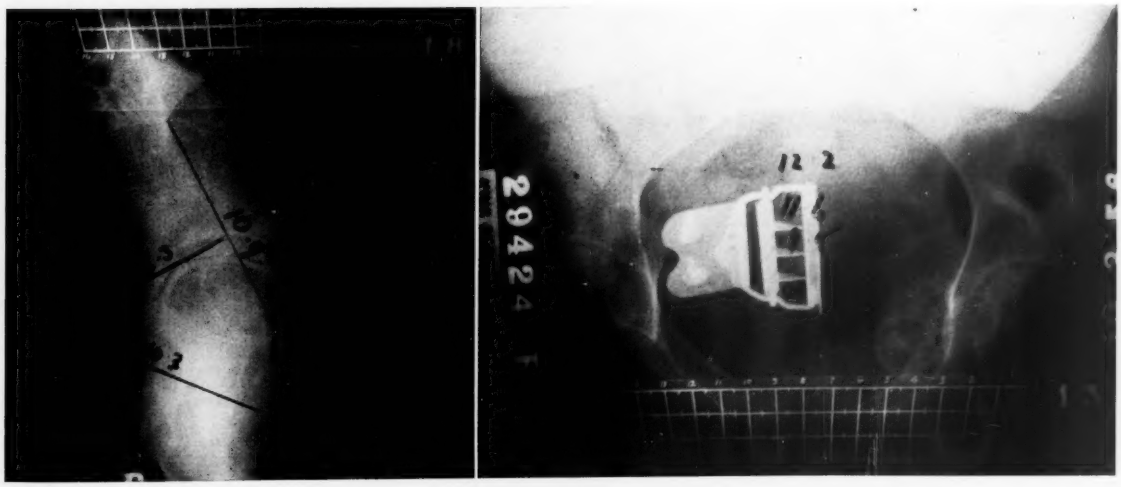


FIGURE III.

X-ray pictures of pelvis showing ossified portions of osteochondroma.

ILLUSTRATIONS TO THE ARTICLE BY J. R. S. DOUGLAS.